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SESSION RESUMED IN FILE 'MEDLINE, CAPLUS, LIFESCI, EMBASE, USPATFULL, BIOSIS'
AT 19:00:23 ON 23 APR 2003
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	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	97.43	97.64

=> s (p70 s6 kinase) or sk6 or (p70()p85 s6 kinase) or (p70()p85 ribosomal s6
kinase) or pp70s6k

L14 2423 (P70 S6 KINASE) OR SK6 OR (P70(W) P85 S6 KINASE) OR (P70(W) P85
RIBOSOMAL S6 KINASE) OR PP70S6K

=> s (p70(s) s6 (s)kinase) or sk6 or (p70()p85(s) s6(s) kinase) or (p70()p85(s)
ribosomal(s) s6(s) kinase) or pp70s6k

L15 2976 (P70(S) S6 (S) KINASE) OR SK6 OR (P70(W) P85(S) S6(S) KINASE)
OR (P70(W) P85(S) RIBOSOMAL(S) S6(S) KINASE) OR PP70S6K

=> s (p70(s) s6 (s)kinase) or sk6 or pp70s6k

L16 2976 (P70(S) S6 (S) KINASE) OR SK6 OR PP70S6K

=> s sk6 or pp70s6k

L17 559 SK6 OR PP70S6K

=> s (p70(s) s6 (s)kinase)

L18 2581 (P70(S) S6 (S) KINASE)

=> s (p70(s)s6(s)kinase)

L19 2581 (P70(S) S6(S) KINASE)

=> s l14 and antisense

L20 73 L14 AND ANTISENSE

=> dup rem l20

PROCESSING COMPLETED FOR L20

L21 52 DUP REM L20 (21 DUPLICATES REMOVED)

=> d l21 ibib abs tot

L21 ANSWER 1 OF 52 CAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER: 2003:117953 CAPLUS

DOCUMENT NUMBER: 138:180755

TITLE: **Antisense oligonucleotides modulation of
p70 S6 kinase expression
for treatment of diseases**

INVENTOR(S): Monia, Brett P.; Cowsert, Lex M.

PATENT ASSIGNEE(S): Isis Pharmaceuticals, Inc., USA

SOURCE: PCT Int. Appl., 93 pp.

CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003012032	A2	20030213	WO 2002-US23123	20020719
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				

PRIORITY APPLN. INFO.: US 2001-920677 A 20010801

AB **Antisense** compds., compns. and methods are provided for modulating the expression of **p70 S6 kinase**. The compns. comprise **antisense** compds., particularly **antisense** oligonucleotides, targeted to nucleic acids encoding **p70 S6 kinase**. Methods of using these compds. for modulation of **p70 S6 kinase** expression and for treatment of diseases assocd. with expression of **p70 S6 kinase** are provided.

L21 ANSWER 2 OF 52 USPATFULL

ACCESSION NUMBER: 2003:100088 USPATFULL
 TITLE: Treatment methods based on microcompetition for a limiting GABP complex
 INVENTOR(S): Polansky, Hanan, Rochester, NY, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003069199	A1	20030410
APPLICATION INFO.:	US 2002-219334	A1	20020815 (10)
RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 2000-732360, filed on 7 Dec 2000, PENDING		
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	APPLICATION		
LEGAL REPRESENTATIVE:	Hanan Polansky, 3159 S. Winton Rd., Rochester, NY, 14623		
NUMBER OF CLAIMS:	26		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	28 Drawing Page(s)		
LINE COUNT:	14837		

AB Microcompetition for GABP between a foreign polynucleotide and a cellular GABP regulated gene is a risk factor associated with chronic disease such as obesity, cancer, atherosclerosis, stroke, osteoarthritis, diabetes, asthma, and other autoimmune diseases. The invention uses this novel discovery to present methods for the treatment of these chronic diseases. The methods are based on modifying such microcompetition, or the effect of such microcompetition on the cell. For instance, treatment may modify the cellular copy number of the foreign polynucleotide, change the rate of complex formation between GABP and either the foreign polynucleotide or the cellular GABP regulated gene, vary the expression of the cellular GABP regulated gene, or manipulate the activity of the gene product of the cellular GABP regulated gene. The invention also presents methods for treatment of chronic diseases resulting from other foreign polynucleotide-type disruptions.

L21 ANSWER 3 OF 52 USPATFULL

ACCESSION NUMBER: 2003:99511 USPATFULL
 TITLE: Drug discovery assays based on microcompetition for a

limiting GABP complex
INVENTOR(S): Polansky, Hanan, Rochester, NY, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003068616	A1	20030410
APPLICATION INFO.:	US 2002-223050	A1	20020814 (10)
RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 2000-732360, filed on 7 Dec 2000, PENDING		
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	APPLICATION		
LEGAL REPRESENTATIVE:	Hanan Polansky, 3159 S. Winton Rd., Rochester, NY, 14623		
NUMBER OF CLAIMS:	55		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	28 Drawing Page(s)		
LINE COUNT:	14981		

AB A recent discovery showed that microcompetition for GABP between a foreign polynucleotide and a cellular GABP regulated gene is a risk factor for some of the major chronic diseases, such as obesity, cancer, atherosclerosis, stroke, osteoarthritis, diabetes, asthma, and other autoimmune diseases. The invention uses this novel discovery to present assays for screening compounds based on their effectiveness in modulating such microcompetition, or the effects of such microcompetition on the cell. The selected compounds can be used in treatment of these chronic diseases. The invention also presents assays for screening compounds that can be used in treatment of chronic diseases resulting from other foreign polynucleotide-type disruptions.

L21 ANSWER 4 OF 52 USPATFULL

ACCESSION NUMBER: 2003:17886 USPATFULL
TITLE: P27 prevents cellular migration
INVENTOR(S): Marks, Andrew R., Larchmont, NY, UNITED STATES
Marx, Steven O., New York, NY, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003013638	A1	20030116
APPLICATION INFO.:	US 2002-172027	A1	20020614 (10)
RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 2001-766944, filed on 22 Jan 2001, PENDING		
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	APPLICATION		
LEGAL REPRESENTATIVE:	Cooper & Dunham LLP, 1185 Avenue of the Americas, New York, NY, 10036		
NUMBER OF CLAIMS:	31		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	5 Drawing Page(s)		
LINE COUNT:	1382		

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB This invention provides methods of preventing cellular migration and of treating cardiovascular diseases and tumor metastasis by increasing the intracellular concentration of cyclin-dependent kinase inhibitor p27 or C3 exoenzyme or by decreasing the intracellular concentration of Rho-kinase, and methods of identifying chemical compounds for use in such treatments.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L21 ANSWER 5 OF 52 USPATFULL

ACCESSION NUMBER: 2003:95980 USPATFULL
TITLE: Reverse transfection method
INVENTOR(S): Sabatini, David M., Cambridge, MA, United States

PATENT ASSIGNEE(S): Whitehead Institute for Biomedical Research, Cambridge,
MA, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6544790	B1	20030408
APPLICATION INFO.:	US 2000-664297		20000918 (9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-193580P	20000330 (60)
	US 1999-154737P	19990917 (60)
DOCUMENT TYPE:	Utility	
FILE SEGMENT:	GRANTED	
PRIMARY EXAMINER:	Yucel, Remy	
ASSISTANT EXAMINER:	Katcheves, Konstantina	
LEGAL REPRESENTATIVE:	Clark & Elbing LLP, Bieker-Brady, Kristina	
NUMBER OF CLAIMS:	151	
EXEMPLARY CLAIM:	1	
NUMBER OF DRAWINGS:	13 Drawing Figure(s); 6 Drawing Page(s)	
LINE COUNT:	1525	

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB A reverse transfection method of introducing DNA of interest into cells
and arrays, including microarrays, of reverse transfected cells.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L21 ANSWER 6 OF 52 MEDLINE DUPLICATE 1
ACCESSION NUMBER: 2003047472 MEDLINE
DOCUMENT NUMBER: 22444672 PubMed ID: 12556481
TITLE: SKIP negatively regulates insulin-induced GLUT4
translocation and membrane ruffle formation.
AUTHOR: Ijuin Takeshi; Takenawa Tadaomi
CORPORATE SOURCE: Department of Biochemistry, Institute of Medical Science,
University of Tokyo, Minato-ku, Tokyo 108-8639, Japan.
SOURCE: MOLECULAR AND CELLULAR BIOLOGY, (2003 Feb) 23 (4) 1209-20.
Journal code: 8109087. ISSN: 0270-7306.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200303
ENTRY DATE: Entered STN: 20030131
Last Updated on STN: 20030306
Entered Medline: 20030305

AB Skeletal muscle and kidney enriched inositol phosphatase (SKIP) is an
inositol polyphosphate 5-phosphatase that hydrolyzes phosphatidylinositol
3,4,5-trisphosphate [PI(3,4,5)P3] to downregulate intracellular levels.
In this study, we show that SKIP inhibits phosphoinositide 3-kinase
signaling in insulin-stimulated CHO cells. Ectopic expression of SKIP did
not inhibit insulin-induced PI(3,4,5)P3 generation but did rapidly
decrease insulin-induced intracellular PI(3,4,5)P3 levels compared with
those in control cells. Further, insulin-induced phosphorylation of some
downstream targets such as Akt and p70 S6
kinase was markedly inhibited by the ectopic expression of SKIP,
whereas phosphorylation of mitogen-activated protein kinase was not. In
contrast, downregulation of intracellular SKIP levels by antisense
oligonucleotides dramatically enhanced Akt (protein kinase B)
phosphorylation in response to insulin, suggesting that endogenous SKIP
downregulates insulin signaling. SKIP also markedly inhibited GLUT4
translocation and membrane ruffle formation. We conclude that SKIP
preferentially regulates glucose transport and actin cytoskeletal
rearrangement among a variety of PI(3,4,5)P3 downstream events.

L21 ANSWER 7 OF 52 CAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER: 2002:772401 CAPLUS
DOCUMENT NUMBER: 137:258500
TITLE: Protein and cDNA sequences of human p70 ribosomal S6
kinase 13.97 and therapeutical uses
INVENTOR(S): Mao, Yumin; Xie, Yi
PATENT ASSIGNEE(S): Bode Gene Development Co., Ltd., Shanghai, Peop. Rep.
China
SOURCE: Faming Zhuanli Shenqing Gongkai Shuomingshu, 34 pp.
CODEN: CNXXEV
DOCUMENT TYPE: Patent
LANGUAGE: Chinese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
CN 1331307	A	20020116	CN 2000-116750	20000626

PRIORITY APPLN. INFO.: CN 2000-116750 20000626

AB The invention provides the protein and cDNA sequences of a novel human p70 ribosomal S6 kinase 13.97 with the mol. wt. of 14 kilodaltons cloned from human fetal brain. In particular, the invention discloses that the gene encoding this protein has a similar gene expression pattern with gene encoding **p70 S6 kinase**. The invention also relates to construction of p70 ribosomal S6 kinase 13.97 expression vector for prepn. of recombinant protein using prokaryotes or eukaryotes. The invention relates to prepn. of antibody against this protein. The invention further relates to the PCR primers, nucleic acid probes, DNA fragments and protein agonists or antagonists specific for this gene or gene product for the diagnosis as well as treatment of various diseases, such as malignant tumors, blood diseases, development disorders, HIV infection, immune disorders or inflammations.

L21 ANSWER 8 OF 52 USPATFULL

ACCESSION NUMBER: 2002:280045 USPATFULL
TITLE: Expression cloning method
INVENTOR(S): Bogan, Jonathan S., Belmont, MA, UNITED STATES
Lodish, Harvey F., Brookline, MA, UNITED STATES
PATENT ASSIGNEE(S): Whitehead Institute for Biomedical Research, Cambridge, MA (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002155479	A1	20021024
APPLICATION INFO.:	US 2002-58820	A1	20020128 (10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2001-264816P	20010126 (60)
	US 2001-325651P	20010928 (60)
	US 1999-138237P	19990609 (60)
	US 1999-154078P	19990915 (60)
DOCUMENT TYPE:	Utility	
FILE SEGMENT:	APPLICATION	
LEGAL REPRESENTATIVE:	HAMILTON, BROOK, SMITH & REYNOLDS, P.C., 530 VIRGINIA ROAD, P.O. BOX 9133, CONCORD, MA, 01742-9133	
NUMBER OF CLAIMS:	57	
EXEMPLARY CLAIM:	1	
NUMBER OF DRAWINGS:	43 Drawing Page(s)	
LINE COUNT:	2585	

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Described herein is a method of expression cloning useful for identifying and obtaining proteins involved in GLUT4 trafficking in mammalian cells and, thus, in insulin-stimulated glucose uptake by such

cells. In particular, an enrichment strategy for expression cloning proteins involved in GLUT4 trafficking at the plasma membrane is described. Proteins identified by the method and uses therefore are also described.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L21 ANSWER 9 OF 52 USPATFULL

ACCESSION NUMBER: 2002:272900 USPATFULL
 TITLE: Stimulus-inducible protein kinase complex and methods of use therefor
 INVENTOR(S): Mercurio, Frank, San Diego, CA, UNITED STATES
 Zhu, Hengyi, San Diego, CA, UNITED STATES
 Barbosa, Miguel, San Diego, CA, UNITED STATES
 Li, Jian Wu, San Diego, CA, UNITED STATES
 Murray, Brion W., San Diego, CA, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002151021	A1	20021017
APPLICATION INFO.:	US 2001-844908	A1	20010427 (9)
RELATED APPLN. INFO.:	Division of Ser. No. US 1997-910820, filed on 13 Aug 1997, PATENTED Continuation-in-part of Ser. No. US 1996-697393, filed on 26 Aug 1996, PATENTED		
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	APPLICATION		
LEGAL REPRESENTATIVE:	Timothy X. Gibson, Mathews, Collins, Shepherd & Gould, Suite 306, 100 Thanet Circle, Princeton, NJ, 08540		
NUMBER OF CLAIMS:	32		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	31 Drawing Page(s)		
LINE COUNT:	2343		

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Compositions and methods are provided for treating NF-.kappa.B-related conditions. In particular, the invention provides a stimulus-inducible IKK signalsome, and components and variants thereof. An IKK signalsome or component thereof may be used, for example, to identify antibodies and other modulating agents that inhibit or activate signal transduction via the NF-.kappa.B cascade. IKK signalsome, components thereof and/or modulating agents may also be used for the treatment of diseases associated with NF-.kappa.B activation.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L21 ANSWER 10 OF 52 USPATFULL

ACCESSION NUMBER: 2002:258894 USPATFULL
 TITLE: 38646, a novel guanine nucleotide exchange factor and uses therefor
 INVENTOR(S): Glucksmann, Maria Alexandra, Lexington, MA, UNITED STATES
 PATENT ASSIGNEE(S): Millennium Pharmaceuticals, Inc., Cambridge, MA (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002142464	A1	20021003
APPLICATION INFO.:	US 2001-950491	A1	20010910 (9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-231089P	20000908 (60)
DOCUMENT TYPE:	Utility	
FILE SEGMENT:	APPLICATION	
LEGAL REPRESENTATIVE:	AKIN, GUMP, STRAUSS, HAUER & FELD, L.L.P., ONE COMMERCE	

SQUARE, 2005 MARKET STREET, SUITE 2200, PHILADELPHIA,
PA, 19103

NUMBER OF CLAIMS: 22
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 15 Drawing Page(s)
LINE COUNT: 4625

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The invention provides isolated nucleic acids molecules, designated 38646 nucleic acid molecules, which encode a novel guanine-nucleotide exchange factor. The invention also provides **antisense** nucleic acid molecules, recombinant expression vectors containing 38646 nucleic acid molecules, host cells into which the expression vectors have been introduced, and non-human transgenic animals in which a 38646 gene has been introduced or disrupted. The invention still further provides isolated 38646 proteins, fusion proteins, antigenic peptides and anti-38646 antibodies. Diagnostic methods utilizing compositions of the invention are also provided. 38646 expression and activity can be modulated to affect cell shape, motility, cytoskeleton organization, and intracellular protein and vesicle localization or to affect the tensile strength or integrity of a tissue.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L21 ANSWER 11 OF 52 USPATFULL

ACCESSION NUMBER: 2002:251221 USPATFULL
TITLE: ASIP-related proteins
INVENTOR(S): Reddy, Roopa, Sunnyvale, CA, UNITED STATES
Tang, Y. Tom, San Jose, CA, UNITED STATES
Baughn, Mariah R., San Leandro, CA, UNITED STATES
Krasnow, Randi E., Stanford, CA, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002137166	A1	20020926
APPLICATION INFO.:	US 2001-757781	A1	20010109 (9)
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	APPLICATION		
LEGAL REPRESENTATIVE:	INCYTE GENOMICS, INC., 3160 PORTER DRIVE, PALO ALTO, CA, 94304		
NUMBER OF CLAIMS:	22		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	40 Drawing Page(s)		
LINE COUNT:	3608		

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The invention provides cDNAs which encode ASIP-related proteins. It also provides for the use of the cDNAs, fragments, complements, and variants thereof and of the encoded proteins, portions thereof and antibodies thereto for diagnosis and treatment of cancer, particularly bladder transitional cell carcinoma. The invention additionally provides expression vectors and host cells for the production of the protein and a transgenic model system.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L21 ANSWER 12 OF 52 USPATFULL

ACCESSION NUMBER: 2002:243110 USPATFULL
TITLE: Human Ste20-like stress activated serine/threonine kinase
INVENTOR(S): Moore, William Craig, West Grove, PA, UNITED STATES
Norris, Tyrrell Errick, New Castle, DE, UNITED STATES
Silberstein, David Shay, Kennett Square, PA, UNITED STATES

NUMBER	KIND	DATE
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PATENT INFORMATION: US 2002132296 A1 20020919
 APPLICATION INFO.: US 2001-906397 A1 20010716 (9)
 RELATED APPLN. INFO.: Division of Ser. No. US 1998-152406, filed on 14 Sep 1998, GRANTED, Pat. No. US 6265560

	NUMBER	DATE
PRIORITY INFORMATION:	GB 1997-19920	19970919
DOCUMENT TYPE:	Utility	
FILE SEGMENT:	APPLICATION	
LEGAL REPRESENTATIVE:	ASTRA ZENECA PHARMACEUTICALS LP, GLOBAL INTELLECTUAL PROPERTY, 1800 CONCORD PIKE, WILMINGTON, DE, 19850-5437	
NUMBER OF CLAIMS:	12	
EXEMPLARY CLAIM:	1	
NUMBER OF DRAWINGS:	10 Drawing Page(s)	
LINE COUNT:	2644	

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB A novel human signal-transduction kinase polypeptide is described which is expressed at a particularly high level in human leukocytes. A full length cDNA which encodes the novel stress-activated serine/threonine kinase polypeptide is disclosed as well as the interior structural region and the amino acid residue sequence of the native biological molecule. Methods are provided to identify compounds that modulate the biological activity of the human Ste20-like stress-activated serine/threonine signal transduction kinase.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L21 ANSWER 13 OF 52 USPATFULL
 ACCESSION NUMBER: 2002:186077 USPATFULL
 TITLE: P27 prevents cellular migration
 INVENTOR(S): Marks, Andrew R., Larchmont, NY, UNITED STATES
 Marx, Steven O., New York, NY, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002098998	A1	20020725
APPLICATION INFO.:	US 2001-766944	A1	20010122 (9)
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	APPLICATION		
LEGAL REPRESENTATIVE:	Cooper & Dunham LLP, 1185 Avenue of the Americas, New York, NY, 10036		
NUMBER OF CLAIMS:	24		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	5 Drawing Page(s)		
LINE COUNT:	1006		

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB This invention provides methods of preventing cellular migration and of treating cardiovascular diseases and tumor metastasis by increasing cyclin-dependent kinase inhibitor p27 activity, and methods of identifying chemical compounds for use in such treatments.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L21 ANSWER 14 OF 52 USPATFULL
 ACCESSION NUMBER: 2002:32181 USPATFULL
 TITLE: Methods of monitoring enzyme activity
 INVENTOR(S): Griffiths, Gary, Oldham, UNITED KINGDOM

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002019002	A1	20020214
APPLICATION INFO.:	US 2001-877919	A1	20010607 (9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-211313P	20000613 (60)
DOCUMENT TYPE:	Utility	
FILE SEGMENT:	APPLICATION	
LEGAL REPRESENTATIVE:	PALMER & DODGE, LLP, ONE BEACON STREET, BOSTON, MA, 02108-3190	
NUMBER OF CLAIMS:	40	
EXEMPLARY CLAIM:	1	
LINE COUNT:	3633	

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB We describe a method for monitoring the activity of an enzyme, the method comprising the steps of: providing a binding domain which includes a site for enzymatic modification; providing a binding partner which binds to the binding domain in a manner which is dependent upon modification of the site. The binding domain is contacted with the enzyme; and binding of the binding domain to the binding partner is detected as an indication of the activity of the enzyme. One of the binding domain and binding partner comprises a polypeptide and the other of the binding domain and binding partner comprises a nucleic acid.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L21 ANSWER 15 OF 52 USPATFULL

ACCESSION NUMBER: 2002:27776 USPATFULL
 TITLE: Drug/drug delivery systems for the prevention and treatment of vascular disease
 INVENTOR(S): Falotico, Robert, Belle Mead, NJ, UNITED STATES
 Kopia, Gregory A., Neshanic, NJ, UNITED STATES
 Llanos, Gerard H., Stewartsville, NJ, UNITED STATES
 Siekierka, John, City Towaco, NJ, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002016625	A1	20020207
APPLICATION INFO.:	US 2001-850232	A1	20010507 (9)
RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 2000-575480, filed on 19 May 2000, UNKNOWN		
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	APPLICATION		
LEGAL REPRESENTATIVE:	AUDLEY A. CIAMPORCERO JR., JOHNSON & JOHNSON, ONE JOHNSON & JOHNSON PLAZA, NEW BRUNSWICK, NJ, 08933-7003		
NUMBER OF CLAIMS:	17		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	2 Drawing Page(s)		
LINE COUNT:	918		

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB A drug and drug delivery system may be utilized in the treatment of vascular disease. A local delivery system is coated with rapamycin or other suitable drug, agent or compound and delivered intraluminally for the treatment and prevention of neointimal hyperplasia following percutaneous transluminal coronary angiography. The local delivery of the drugs or agents provides for increased effectiveness and lower systemic toxicity.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L21 ANSWER 16 OF 52 USPATFULL

ACCESSION NUMBER: 2002:12830 USPATFULL
 TITLE: Drug/drug delivery systems for the prevention and treatment of vascular disease
 INVENTOR(S): Falotico, Robert, Belle Mead, NJ, UNITED STATES
 Siekierka, John, Towaco, NJ, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002007215	A1	20020117
APPLICATION INFO.:	US 2001-850365	A1	20010507 (9)
RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 2000-575480, filed on 19 May 2000, UNKNOWN		
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	APPLICATION		
LEGAL REPRESENTATIVE:	AUDLEY A. CIAMPORCERO JR., JOHNSON & JOHNSON, ONE JOHNSON & JOHNSON PLAZA, NEW BRUNSWICK, NJ, 08933-7003		
NUMBER OF CLAIMS:	14		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	2 Drawing Page(s)		
LINE COUNT:	924		

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB A drug and drug delivery system may be utilized in the treatment of vascular disease. A local delivery system is coated with rapamycin or other suitable drug, agent or compound and delivered intraluminally for the treatment and prevention of neointimal hyperplasia following percutaneous transluminal coronary angiography. The local delivery of the drugs or agents provides for increased effectiveness and lower systemic toxicity.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L21 ANSWER 17 OF 52 USPATFULL

ACCESSION NUMBER: 2002:12829 USPATFULL
 TITLE: Drug/drug delivery systems for the prevention and treatment of vascular disease
 INVENTOR(S): Falotico, Robert, Belle Mead, NJ, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002007214	A1	20020117
APPLICATION INFO.:	US 2001-850293	A1	20010507 (9)
RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 2000-575480, filed on 19 May 2000, UNKNOWN		
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	APPLICATION		
LEGAL REPRESENTATIVE:	AUDLEY A. CIAMPORCERO JR., JOHNSON & JOHNSON, ONE JOHNSON & JOHNSON PLAZA, NEW BRUNSWICK, NJ, 08933-7003		
NUMBER OF CLAIMS:	15		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	2 Drawing Page(s)		
LINE COUNT:	916		

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB A drug and drug delivery system may be utilized in the treatment of vascular disease. A local delivery system is coated with rapamycin or other suitable drug, agent or compound and delivered intraluminally for the treatment and prevention of neointimal hyperplasia following percutaneous transluminal coronary angiography. The local delivery of the drugs or agents provides for increased effectiveness and lower systemic toxicity.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L21 ANSWER 18 OF 52 USPATFULL

ACCESSION NUMBER: 2002:12828 USPATFULL
 TITLE: Drug/drug delivery systems for the prevention and treatment of vascular disease
 INVENTOR(S): Falotico, Robert, Belle Mead, NJ, UNITED STATES
 Kopia, Gregory A., Hillsborough, NJ, UNITED STATES
 Llanos, Gerard H., Stewartsville, NJ, UNITED STATES

Sieklerka, John, Towaco, NJ, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002007213	A1	20020117
APPLICATION INFO.:	US 2001-850233	A1	20010507 (9)
RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 2000-575480, filed on 19 May 2000, UNKNOWN		
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	APPLICATION		
LEGAL REPRESENTATIVE:	AUDLEY A. CIAMPORCERO JR., JOHNSON & JOHNSON, ONE JOHNSON & JOHNSON PLAZA, NEW BRUNSWICK, NJ, 08933-7003		
NUMBER OF CLAIMS:	11		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	2 Drawing Page(s)		
LINE COUNT:	895		
CAS INDEXING IS AVAILABLE FOR THIS PATENT.			

AB A drug and drug delivery system may be utilized in the treatment of vascular disease. A local delivery system is coated with rapamycin or other suitable drug, agent or compound and delivered intraluminally for the treatment and prevention of neointimal hyperplasia following percutaneous transluminal coronary angiography. The local delivery of the drugs or agents provides for increased effectiveness and lower systemic toxicity.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L21 ANSWER 19 OF 52 USPATFULL

ACCESSION NUMBER: 2002:12284 USPATFULL
TITLE: Arrayed transfection method and uses related thereto
INVENTOR(S): Sabatini, David M., Cambridge, MA, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002006664	A1	20020117
APPLICATION INFO.:	US 2001-817003	A1	20010322 (9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-193580P	20000330 (60)
	US 1999-154737P	19990917 (60)
DOCUMENT TYPE:	Utility	
FILE SEGMENT:	APPLICATION	
LEGAL REPRESENTATIVE:	ROPES & GRAY, ONE INTERNATIONAL PLACE, BOSTON, MA, 02110-2624	
NUMBER OF CLAIMS:	37	
EXEMPLARY CLAIM:	1	
NUMBER OF DRAWINGS:	7 Drawing Page(s)	
LINE COUNT:	2671	
CAS INDEXING IS AVAILABLE FOR THIS PATENT.		
AB	An arrayed transfection method of introducing nucleic acid of interest into cells.	

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L21 ANSWER 20 OF 52 USPATFULL

ACCESSION NUMBER: 2002:10838 USPATFULL
TITLE: Antiproliferative drug and delivery device
INVENTOR(S): Falotico, Robert, Belle Mead, NJ, UNITED STATES
Kopia, Gregory A., Hillsborough, NJ, UNITED STATES
Llanos, Gerard H., Stewartsville, NJ, UNITED STATES
Siekjerka, John, Towaco, NJ, UNITED STATES

NUMBER	KIND	DATE
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PATENT INFORMATION: US 2002005206 A1 20020117
APPLICATION INFO.: US 2001-850507 A1 20010507 (9)
RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 2000-575480, filed
on 19 May 2000, UNKNOWN
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: AUDLEY A. CIAMPORCERO JR., JOHNSON & JOHNSON, ONE
JOHNSON & JOHNSON PLAZA, NEW BRUNSWICK, NJ, 08933-7003
NUMBER OF CLAIMS: 15
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 2 Drawing Page(s)
LINE COUNT: 921
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB A drug and drug delivery system may be utilized in the treatment of
vascular disease. A local delivery system is coated with rapamycin or
other suitable drug, agent or compound and delivered intraluminally for
the treatment and prevention of neointimal hyperplasia following
percutaneous transluminal coronary angiography. The local delivery of
the drugs or agents provides for increased effectiveness and lower
systemic toxicity.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L21 ANSWER 21 OF 52 USPATFULL

ACCESSION NUMBER: 2002:325826 USPATFULL
TITLE: Mammalian proteins that bind to FKBP12 in a
rapamycin-dependent fashion
INVENTOR(S): Sabatini, David M., Baltimore, MD, United States
Erdjument-Bromage, Hediye, New York, NY, United States
Lui, Mary, Kew Gardens, NY, United States
Tempst, Paul, New York, NY, United States
Snyder, Solomon H., Baltimore, MD, United States
PATENT ASSIGNEE(S): The Johns Hopkins University, Baltimore, MD, United
States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6492106	B1	20021210
APPLICATION INFO.:	US 1994-305790		19940914 (8)
RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 1994-265967, filed on 27 Jun 1994		
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	GRANTED		
PRIMARY EXAMINER:	Patterson, Jr., Charles L.		
ASSISTANT EXAMINER:	Kerr, Kathleen		
LEGAL REPRESENTATIVE:	Banner & Witcoff, Ltd.		
NUMBER OF CLAIMS:	12		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	11 Drawing Figure(s); 10 Drawing Page(s)		
LINE COUNT:	2121		

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB A protein complex containing 245 kDa and 35 kDa components, designated
RAFT1 and RAFT2 (for Rapamycin And FKBP12 Target) interacts with FKBP12
in a rapamycin-dependent manner. This interaction has the
pharmacological characteristics expected from the observed in vivo
effects of rapamycin: it occurs at low nanomolar concentrations of
rapamycin and is competed by excess FK506. Sequences (330 amino acids
total) of tryptic peptides derived from the affinity purified 245 kDa
RAFT1 reveals striking homologies to the predicted products of the yeast
TOR genes, which were originally identified by mutations that confer
rapamycin resistance in yeast. A RAFT1 cDNA was obtained and found to
encode a 289 kDa protein (2550 amino acids) that is 43% and 39%
identical to TOR2 and TOR1, respectively.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L21 ANSWER 22 OF 52 USPATFULL

ACCESSION NUMBER: 2002:303872 USPATFULL

TITLE: Lipid kinase

INVENTOR(S): Vanhaesebroeck, Bart, London, UNITED KINGDOM
Waterfield, Michael Derek, London, UNITED KINGDOM

PATENT ASSIGNEE(S): Ludwig Institute for Cancer Research, New York, NY,
United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6482623	B1	20021119
	WO 9746688		19971211
APPLICATION INFO.:	US 1998-194640		19981201 (9)
	WO 1997-GB1471		19970530
			19981201 PCT 371 date

	NUMBER	DATE
PRIORITY INFORMATION:	GB 1996-11460	19960601
DOCUMENT TYPE:	Utility	
FILE SEGMENT:	GRANTED	
PRIMARY EXAMINER:	Achutamurthy, Ponnathapu	
ASSISTANT EXAMINER:	Rao, Manjunath N.	
LEGAL REPRESENTATIVE:	Fulbright & Jaworski LLP	
NUMBER OF CLAIMS:	20	
EXEMPLARY CLAIM:	1	
NUMBER OF DRAWINGS:	15 Drawing Figure(s); 18 Drawing Page(s)	
LINE COUNT:	1568	

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The invention relates to a novel lipid kinase which is part of the PI3 Kinase family. PI3 Kinases catalyze the addition of phosphate to inositol generating inositol mono, di and triphosphate. Inositol phosphates have been implicated in regulating intracellular signalling cascades resulting in alternations in gene expression which, amongst other effects, can result in cytoskeletal remodelling and modulation of cellular motility. More particularly the invention relates to a novel

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=> s (p70 s6 kinase) or sk6 or (p70())p85 s6 kinase) or (p70())p85 ribosomal s6
kinase) or pp70s6k

L14 2423 (P70 S6 KINASE) OR SK6 OR (P70(W) P85 S6 KINASE) OR (P70(W) P85
RIBOSOMAL S6 KINASE) OR PP70S6K

=> s (p70(s) s6 (s)kinase) or sk6 or (p70())p85(s) s6(s) kinase) or (p70())p85(s)
ribosomal(s) s6(s) kinase) or pp70s6k

L15 2976 (P70(S) S6 (S) KINASE) OR SK6 OR (P70(W) P85(S) S6(S) KINASE)
OR (P70(W) P85(S) RIBOSOMAL(S) S6(S) KINASE) OR PP70S6K

=> s (p70(s) s6 (s)kinase) or sk6 or pp70s6k

L16 2976 (P70(S) S6 (S) KINASE) OR SK6 OR PP70S6K

=> s sk6 or pp70s6k

L17 559 SK6 OR PP70S6K

=> s (p70(s) s6 (s)kinase)

L18 2581 (P70(S) S6 (S) KINASE)

=> s (p70(s)s6(s)kinase)

L19 2581 (P70(S) S6(S) KINASE)

=> s l14 and antisense

L20 73 L14 AND ANTISENSE

=> dup rem l20

PROCESSING COMPLETED FOR L20

L21 52 DUP REM L20 (21 DUPLICATES REMOVED)

=> d l21 ibib abs tot

L21 ANSWER 1 OF 52 CAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER: 2003:117953 CAPLUS

DOCUMENT NUMBER: 138:180755

TITLE: **Antisense oligonucleotides modulation of p70 S6 kinase expression for treatment of diseases**

INVENTOR(S): Monia, Brett P.; Cowser, Lex M.

PATENT ASSIGNEE(S): Isis Pharmaceuticals, Inc., USA

SOURCE: PCT Int. Appl., 93 pp.
CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003012032	A2	20030213	WO 2002-US23123	20020719
<p>W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM</p> <p>RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG</p>				

PRIORITY APPLN. INFO.: US 2001-920677 A 20010801

AB **Antisense** compds., compns. and methods are provided for modulating the expression of **p70 S6 kinase**. The compns. comprise **antisense** compds., particularly **antisense** oligonucleotides, targeted to nucleic acids encoding **p70 S6 kinase**. Methods of using these compds. for modulation of **p70 S6 kinase** expression and for treatment of diseases assocd. with expression of **p70 S6 kinase** are provided.

L21 ANSWER 2 OF 52 USPATFULL

ACCESSION NUMBER: 2003:100088 USPATFULL

TITLE: Treatment methods based on microcompetition for a limiting GABP complex

INVENTOR(S): Polansky, Hanan, Rochester, NY, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003069199	A1	20030410
APPLICATION INFO.:	US 2002-219334	A1	20020815 (10)
RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 2000-732360, filed on 7 Dec 2000, PENDING		
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	APPLICATION		
LEGAL REPRESENTATIVE:	Hanan Polansky, 3159 S. Winton Rd., Rochester, NY, 14623		
NUMBER OF CLAIMS:	26		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	28 Drawing Page(s)		
LINE COUNT:	14837		

AB Microcompetition for GABP between a foreign polynucleotide and a cellular GABP regulated gene is a risk factor associated with chronic disease such as obesity, cancer, atherosclerosis, stroke, osteoarthritis, diabetes, asthma, and other autoimmune diseases. The invention uses this novel discovery to present methods for the treatment of these chronic diseases. The methods are based on modifying such microcompetition, or the effect of such microcompetition on the cell. For instance, treatment may modify the cellular copy number of the

foreign polynucleotide, change the rate of complex formation between GABP and either the foreign polynucleotide or the cellular GABP regulated gene, vary the expression of the cellular GABP regulated gene, or manipulate the activity of the gene product of the cellular GABP regulated gene. The invention also presents methods for treatment of chronic diseases resulting from other foreign polynucleotide-type disruptions.

L21 ANSWER 3 OF 52 USPATFULL

ACCESSION NUMBER: 2003:99511 USPATFULL
 TITLE: Drug discovery assays based on microcompetition for a limiting GABP complex
 INVENTOR(S): Polansky, Hanan, Rochester, NY, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003068616	A1	20030410
APPLICATION INFO.:	US 2002-223050	A1	20020814 (10)
RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 2000-732360, filed on 7 Dec 2000, PENDING		
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	APPLICATION		
LEGAL REPRESENTATIVE:	Hanan Polansky, 3159 S. Winton Rd., Rochester, NY, 14623		
NUMBER OF CLAIMS:	55		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	28 Drawing Page(s)		
LINE COUNT:	14981		

AB A recent discovery showed that microcompetition for GABP between a foreign polynucleotide and a cellular GABP regulated gene is a risk factor for some of the major chronic diseases, such as obesity, cancer, atherosclerosis, stroke, osteoarthritis, diabetes, asthma, and other autoimmune diseases. The invention uses this novel discovery to present assays for screening compounds based on their effectiveness in modulating such microcompetition, or the effects of such microcompetition on the cell. The selected compounds can be used in treatment of these chronic diseases. The invention also presents assays for screening compounds that can be used in treatment of chronic diseases resulting from other foreign polynucleotide-type disruptions.

L21 ANSWER 4 OF 52 USPATFULL

ACCESSION NUMBER: 2003:17886 USPATFULL
 TITLE: P27 prevents cellular migration
 INVENTOR(S): Marks, Andrew R., Larchmont, NY, UNITED STATES
 Marx, Steven O., New York, NY, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003013638	A1	20030116
APPLICATION INFO.:	US 2002-172027	A1	20020614 (10)
RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 2001-766944, filed on 22 Jan 2001, PENDING		
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	APPLICATION		
LEGAL REPRESENTATIVE:	Cooper & Dunham LLP, 1185 Avenue of the Americas, New York, NY, 10036		
NUMBER OF CLAIMS:	31		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	5 Drawing Page(s)		
LINE COUNT:	1382		

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB This invention provides methods of preventing cellular migration and of

treating cardiovascular diseases and tumor metastasis by increasing the intracellular concentration of cyclin-dependent kinase inhibitor p27 or C3 exoenzyme or by decreasing the intracellular concentration of Rho-kinase, and methods of identifying chemical compounds for use in such treatments.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L21 ANSWER 5 OF 52 USPATFULL

ACCESSION NUMBER: 2003:95980 USPATFULL
TITLE: Reverse transfection method
INVENTOR(S): Sabatini, David M., Cambridge, MA, United States
PATENT ASSIGNEE(S): Whitehead Institute for Biomedical Research, Cambridge, MA, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6544790	B1	20030408
APPLICATION INFO.:	US 2000-664297		20000918 (9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-193580P	20000330 (60)
	US 1999-154737P	19990917 (60)
DOCUMENT TYPE:	Utility	
FILE SEGMENT:	GRANTED	
PRIMARY EXAMINER:	Yucel, Remy	
ASSISTANT EXAMINER:	Katcheves, Konstantina	
LEGAL REPRESENTATIVE:	Clark & Elbing LLP, Bieker-Brady, Kristina	
NUMBER OF CLAIMS:	151	
EXEMPLARY CLAIM:	1	
NUMBER OF DRAWINGS:	13 Drawing Figure(s); 6 Drawing Page(s)	
LINE COUNT:	1525	

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB A reverse transfection method of introducing DNA of interest into cells and arrays, including microarrays, of reverse transfected cells.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L21 ANSWER 6 OF 52 MEDLINE DUPLICATE 1

ACCESSION NUMBER: 2003047472 MEDLINE
DOCUMENT NUMBER: 22444672 PubMed ID: 12556481
TITLE: SKIP negatively regulates insulin-induced GLUT4 translocation and membrane ruffle formation.
AUTHOR: Ijuin Takeshi; Takenawa Tadaomi
CORPORATE SOURCE: Department of Biochemistry, Institute of Medical Science, University of Tokyo, Minato-ku, Tokyo 108-8639, Japan.
SOURCE: MOLECULAR AND CELLULAR BIOLOGY, (2003 Feb) 23 (4) 1209-20. Journal code: 8109087. ISSN: 0270-7306.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200303
ENTRY DATE: Entered STN: 20030131
Last Updated on STN: 20030306
Entered Medline: 20030305

AB Skeletal muscle and kidney enriched inositol phosphatase (SKIP) is an inositol polyphosphate 5-phosphatase that hydrolyzes phosphatidylinositol 3,4,5-trisphosphate [PI(3,4,5)P3] to downregulate intracellular levels. In this study, we show that SKIP inhibits phosphoinositide 3-kinase signaling in insulin-stimulated CHO cells. Ectopic expression of SKIP did not inhibit insulin-induced PI(3,4,5)P3 generation but did rapidly decrease insulin-induced intracellular PI(3,4,5)P3 levels compared with

those in control cells. Further, insulin-induced phosphorylation of some downstream targets such as Akt and **p70 S6 kinase** was markedly inhibited by the ectopic expression of SKIP, whereas phosphorylation of mitogen-activated protein kinase was not. In contrast, downregulation of intracellular SKIP levels by **antisense** oligonucleotides dramatically enhanced Akt (protein kinase B) phosphorylation in response to insulin, suggesting that endogenous SKIP downregulates insulin signaling. SKIP also markedly inhibited GLUT4 translocation and membrane ruffle formation. We conclude that SKIP preferentially regulates glucose transport and actin cytoskeletal rearrangement among a variety of PI(3,4,5)P3 downstream events.

L21 ANSWER 7 OF 52 CAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER: 2002:772401 CAPLUS
DOCUMENT NUMBER: 137:258500
TITLE: Protein and cDNA sequences of human p70 ribosomal S6 kinase 13.97 and therapeutical uses
INVENTOR(S): Mao, Yumin; Xie, Yi
PATENT ASSIGNEE(S): Bode Gene Development Co., Ltd., Shanghai, Peop. Rep. China
SOURCE: Faming Zhuanli Shenqing Gongkai Shuomingshu, 34 pp.
CODEN: CNXXEV
DOCUMENT TYPE: Patent
LANGUAGE: Chinese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
CN 1331307	A	20020116	CN 2000-116750	20000626
PRIORITY APPLN. INFO.:			CN 2000-116750	20000626

AB The invention provides the protein and cDNA sequences of a novel human p70 ribosomal S6 kinase 13.97 with the mol. wt. of 14 kilodaltons cloned from human fetal brain. In particular, the invention discloses that the gene encoding this protein has a similar gene expression pattern with gene encoding **p70 S6 kinase**. The invention also relates to construction of p70 ribosomal S6 kinase 13.97 expression vector for prepn. of recombinant protein using prokaryotes or eukaryotes. The invention relates to prepn. of antibody against this protein. The invention further relates to the PCR primers, nucleic acid probes, DNA fragments and protein agonists or antagonists specific for this gene or gene product for the diagnosis as well as treatment of various diseases, such as malignant tumors, blood diseases, development disorders, HIV infection, immune disorders or inflammations.

L21 ANSWER 8 OF 52 USPATFULL

ACCESSION NUMBER: 2002:280045 USPATFULL
TITLE: Expression cloning method
INVENTOR(S): Bogan, Jonathan S., Belmont, MA, UNITED STATES
Lodish, Harvey F., Brookline, MA, UNITED STATES
PATENT ASSIGNEE(S): Whitehead Institute for Biomedical Research, Cambridge, MA (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002155479	A1	20021024
APPLICATION INFO.:	US 2002-58820	A1	20020128 (10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2001-264816P	20010126 (60)
	US 2001-325651P	20010928 (60)
	US 1999-138237P	19990609 (60)
	US 1999-154078P	19990915 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: HAMILTON, BROOK, SMITH & REYNOLDS, P.C., 530 VIRGINIA
ROAD, P.O. BOX 9133, CONCORD, MA, 01742-9133
NUMBER OF CLAIMS: 57
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 43 Drawing Page(s)
LINE COUNT: 2585

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Described herein is a method of expression cloning useful for identifying and obtaining proteins involved in GLUT4 trafficking in mammalian cells and, thus, in insulin-stimulated glucose uptake by such cells. In particular, an enrichment strategy for expression cloning proteins involved in GLUT4 trafficking at the plasma membrane is described. Proteins identified by the method and uses therefore are also described.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L21 ANSWER 9 OF 52 USPATFULL

ACCESSION NUMBER: 2002:272900 USPATFULL
TITLE: Stimulus-inducible protein kinase complex and methods of use therefor
INVENTOR(S): Mercurio, Frank, San Diego, CA, UNITED STATES
Zhu, Hengyi, San Diego, CA, UNITED STATES
Barbosa, Miguel, San Diego, CA, UNITED STATES
Li, Jian Wu, San Diego, CA, UNITED STATES
Murray, Brion W., San Diego, CA, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002151021	A1	20021017
APPLICATION INFO.:	US 2001-844908	A1	20010427 (9)
RELATED APPLN. INFO.:	Division of Ser. No. US 1997-910820, filed on 13 Aug 1997, PATENTED Continuation-in-part of Ser. No. US 1996-697393, filed on 26 Aug 1996, PATENTED		
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	APPLICATION		
LEGAL REPRESENTATIVE:	Timothy X. Gibson, Mathews, Collins, Shepherd & Gould, Suite 306, 100 Thanet Circle, Princeton, NJ, 08540		
NUMBER OF CLAIMS:	32		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	31 Drawing Page(s)		
LINE COUNT:	2343		

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Compositions and methods are provided for treating NF-.kappa.B-related conditions. In particular, the invention provides a stimulus-inducible IKK signalsome, and components and variants thereof. An IKK signalsome or component thereof may be used, for example, to identify antibodies and other modulating agents that inhibit or activate signal transduction via the NF-.kappa.B cascade. IKK signalsome, components thereof and/or modulating agents may also be used for the treatment of diseases associated with NF-.kappa.B activation.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L21 ANSWER 10 OF 52 USPATFULL

ACCESSION NUMBER: 2002:258894 USPATFULL
TITLE: 38646, a novel guanine nucleotide exchange factor and uses therefor
INVENTOR(S): Glucksmann, Maria Alexandra, Lexington, MA, UNITED STATES
PATENT ASSIGNEE(S): Millennium Pharmaceuticals, Inc., Cambridge, MA (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002142464	A1	20021003
APPLICATION INFO.:	US 2001-950491	A1	20010910 (9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-231089P	20000908 (60)
DOCUMENT TYPE:	Utility	
FILE SEGMENT:	APPLICATION	
LEGAL REPRESENTATIVE:	AKIN, GUMP, STRAUSS, HAUER & FELD, L.L.P., ONE COMMERCE SQUARE, 2005 MARKET STREET, SUITE 2200, PHILADELPHIA, PA, 19103	
NUMBER OF CLAIMS:	22	
EXEMPLARY CLAIM:	1	
NUMBER OF DRAWINGS:	15 Drawing Page(s)	
LINE COUNT:	4625	

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The invention provides isolated nucleic acids molecules, designated 38646 nucleic acid molecules, which encode a novel guanine-nucleotide exchange factor. The invention also provides **antisense** nucleic acid molecules, recombinant expression vectors containing 38646 nucleic acid molecules, host cells into which the expression vectors have been introduced, and non-human transgenic animals in which a 38646 gene has been introduced or disrupted. The invention still further provides isolated 38646 proteins, fusion proteins, antigenic peptides and anti-38646 antibodies. Diagnostic methods utilizing compositions of the invention are also provided. 38646 expression and activity can be modulated to affect cell shape, motility, cytoskeleton organization, and intracellular protein and vesicle localization or to affect the tensile strength or integrity of a tissue.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L21 ANSWER 11 OF 52 USPATFULL

ACCESSION NUMBER: 2002:251221 USPATFULL
 TITLE: ASIP-related proteins
 INVENTOR(S): Reddy, Roopa, Sunnyvale, CA, UNITED STATES
 Tang, Y. Tom, San Jose, CA, UNITED STATES
 Baughn, Mariah R., San Leandro, CA, UNITED STATES
 Krasnow, Randi E., Stanford, CA, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002137166	A1	20020926
APPLICATION INFO.:	US 2001-757781	A1	20010109 (9)

DOCUMENT TYPE:	Utility
FILE SEGMENT:	APPLICATION
LEGAL REPRESENTATIVE:	INCYTE GENOMICS, INC., 3160 PORTER DRIVE, PALO ALTO, CA, 94304

NUMBER OF CLAIMS:	22
EXEMPLARY CLAIM:	1
NUMBER OF DRAWINGS:	40 Drawing Page(s)
LINE COUNT:	3608

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The invention provides cDNAs which encode ASIP-related proteins. It also provides for the use of the cDNAs, fragments, complements, and variants thereof and of the encoded proteins, portions thereof and antibodies thereto for diagnosis and treatment of cancer, particularly bladder transitional cell carcinoma. The invention additionally provides expression vectors and host cells for the production of the protein and a transgenic model system.

CAS INDEXING IS AVAILABLE FOR THIS PATENT. '

L21 ANSWER 12 OF 52 USPATFULL

ACCESSION NUMBER: 2002:243110 USPATFULL
TITLE: Human Ste20-like stress activated serine/threonine kinase
INVENTOR(S): Moore, William Craig, West Grove, PA, UNITED STATES
Norris, Tyrrell Errick, New Castle, DE, UNITED STATES
Silberstein, David Shay, Kennett Square, PA, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002132296	A1	20020919
APPLICATION INFO.:	US 2001-906397	A1	20010716 (9)
RELATED APPLN. INFO.:	Division of Ser. No. US 1998-152406, filed on 14 Sep 1998, GRANTED, Pat. No. US 6265560		

	NUMBER	DATE
PRIORITY INFORMATION:	GB 1997-19920	19970919
DOCUMENT TYPE:	Utility	
FILE SEGMENT:	APPLICATION	
LEGAL REPRESENTATIVE:	ASTRA ZENECA PHARMACEUTICALS LP, GLOBAL INTELLECTUAL PROPERTY, 1800 CONCORD PIKE, WILMINGTON, DE, 19850-5437	
NUMBER OF CLAIMS:	12	
EXEMPLARY CLAIM:	1	
NUMBER OF DRAWINGS:	10 Drawing Page(s)	
LINE COUNT:	2644	

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB A novel human signal-transduction kinase polypeptide is described which is expressed at a particularly high level in human leukocytes. A full length cDNA which encodes the novel stress-activated serine/threonine kinase polypeptide is disclosed as well as the interior structural region and the amino acid residue sequence of the native biological molecule. Methods are provided to identify compounds that modulate the biological activity of the human Ste20-like stress-activated serine/threonine signal transduction kinase.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L21 ANSWER 13 OF 52 USPATFULL

ACCESSION NUMBER: 2002:186077 USPATFULL
TITLE: P27 prevents cellular migration
INVENTOR(S): Marks, Andrew R., Larchmont, NY, UNITED STATES
Marx, Steven O., New York, NY, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002098998	A1	20020725
APPLICATION INFO.:	US 2001-766944	A1	20010122 (9)
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	APPLICATION		
LEGAL REPRESENTATIVE:	Cooper & Dunham LLP, 1185 Avenue of the Americas, New York, NY, 10036		
NUMBER OF CLAIMS:	24		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	5 Drawing Page(s)		
LINE COUNT:	1006		

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB This invention provides methods of preventing cellular migration and of treating cardiovascular diseases and tumor metastasis by increasing cyclin-dependent kinase inhibitor p27 activity, and methods of identifying chemical compounds for use in such treatments.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L21 ANSWER 14 OF 52 USPATFULL

ACCESSION NUMBER: 2002:32181 USPATFULL
TITLE: Methods of monitoring enzyme activity
INVENTOR(S): Griffiths, Gary, Oldham, UNITED KINGDOM

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002019002	A1	20020214
APPLICATION INFO.:	US 2001-877919	A1	20010607 (9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-211313P	20000613 (60)
DOCUMENT TYPE:	Utility	
FILE SEGMENT:	APPLICATION	
LEGAL REPRESENTATIVE:	PALMER & DODGE, LLP, ONE BEACON STREET, BOSTON, MA, 02108-3190	
NUMBER OF CLAIMS:	40	
EXEMPLARY CLAIM:	1	
LINE COUNT:	3633	

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB We describe a method for monitoring the activity of an enzyme, the method comprising the steps of: providing a binding domain which includes a site for enzymatic modification; providing a binding partner which binds to the binding domain in a manner which is dependent upon modification of the site. The binding domain is contacted with the enzyme; and binding of the binding domain to the binding partner is detected as an indication of the activity of the enzyme. One of the binding domain and binding partner comprises a polypeptide and the other of the binding domain and binding partner comprises a nucleic acid.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L21 ANSWER 15 OF 52 USPATFULL

ACCESSION NUMBER: 2002:27776 USPATFULL
TITLE: Drug/drug delivery systems for the prevention and treatment of vascular disease
INVENTOR(S): Falotico, Robert, Belle Mead, NJ, UNITED STATES
Kopia, Gregory A., Neshanic, NJ, UNITED STATES
Llanos, Gerard H., Stewartsville, NJ, UNITED STATES
Siekierka, John, City Towaco, NJ, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002016625	A1	20020207
APPLICATION INFO.:	US 2001-850232	A1	20010507 (9)
RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 2000-575480, filed on 19 May 2000, UNKNOWN		
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	APPLICATION		
LEGAL REPRESENTATIVE:	AUDLEY A. CIAMPORCERO JR., JOHNSON & JOHNSON, ONE JOHNSON & JOHNSON PLAZA, NEW BRUNSWICK, NJ, 08933-7003		
NUMBER OF CLAIMS:	17		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	2 Drawing Page(s)		
LINE COUNT:	918		

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB A drug and drug delivery system may be utilized in the treatment of vascular disease. A local delivery system is coated with rapamycin or other suitable drug, agent or compound and delivered intraluminally for the treatment and prevention of neointimal hyperplasia following

percutaneous transluminal coronary angiography. The local delivery of the drugs or agents provides for increased effectiveness and lower systemic toxicity.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L21 ANSWER 16 OF 52 USPATFULL

ACCESSION NUMBER: 2002:12830 USPATFULL

TITLE: Drug/drug delivery systems for the prevention and treatment of vascular disease

INVENTOR(S): Falotico, Robert, Belle Mead, NJ, UNITED STATES
Siekierka, John, Towaco, NJ, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002007215	A1	20020117
APPLICATION INFO.:	US 2001-850365	A1	20010507 (9)
RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 2000-575480, filed on 19 May 2000, UNKNOWN		
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	APPLICATION		
LEGAL REPRESENTATIVE:	AUDLEY A. CIAMPORCERO JR., JOHNSON & JOHNSON, ONE JOHNSON & JOHNSON PLAZA, NEW BRUNSWICK, NJ, 08933-7003		
NUMBER OF CLAIMS:	14		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	2 Drawing Page(s)		
LINE COUNT:	924		

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB A drug and drug delivery system may be utilized in the treatment of vascular disease. A local delivery system is coated with rapamycin or other suitable drug, agent or compound and delivered intraluminally for the treatment and prevention of neointimal hyperplasia following percutaneous transluminal coronary angiography. The local delivery of the drugs or agents provides for increased effectiveness and lower systemic toxicity.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L21 ANSWER 17 OF 52 USPATFULL

ACCESSION NUMBER: 2002:12829 USPATFULL

TITLE: Drug/drug delivery systems for the prevention and treatment of vascular disease

INVENTOR(S): Falotico, Robert, Belle Mead, NJ, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002007214	A1	20020117
APPLICATION INFO.:	US 2001-850293	A1	20010507 (9)
RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 2000-575480, filed on 19 May 2000, UNKNOWN		
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	APPLICATION		
LEGAL REPRESENTATIVE:	AUDLEY A. CIAMPORCERO JR., JOHNSON & JOHNSON, ONE JOHNSON & JOHNSON PLAZA, NEW BRUNSWICK, NJ, 08933-7003		
NUMBER OF CLAIMS:	15		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	2 Drawing Page(s)		
LINE COUNT:	916		

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB A drug and drug delivery system may be utilized in the treatment of vascular disease. A local delivery system is coated with rapamycin or other suitable drug, agent or compound and delivered intraluminally for the treatment and prevention of neointimal hyperplasia following percutaneous transluminal coronary angiography. The local delivery of

the drugs or agents provides for increased effectiveness and lower systemic toxicity.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L21 ANSWER 18 OF 52 USPATFULL

ACCESSION NUMBER: 2002:12828 USPATFULL
TITLE: Drug/drug delivery systems for the prevention and treatment of vascular disease
INVENTOR(S): Falotico, Robert, Belle Mead, NJ, UNITED STATES
Kopia, Gregory A., Hillsborough, NJ, UNITED STATES
Llanos, Gerard H., Stewartsville, NJ, UNITED STATES
Sieklerka, John, Towaco, NJ, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002007213	A1	20020117
APPLICATION INFO.:	US 2001-850233	A1	20010507 (9)
RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 2000-575480, filed on 19 May 2000, UNKNOWN		
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	APPLICATION		
LEGAL REPRESENTATIVE:	AUDLEY A. CIAMPORCERO JR., JOHNSON & JOHNSON, ONE JOHNSON & JOHNSON PLAZA, NEW BRUNSWICK, NJ, 08933-7003		
NUMBER OF CLAIMS:	11		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	2 Drawing Page(s)		
LINE COUNT:	895		

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB A drug and drug delivery system may be utilized in the treatment of vascular disease. A local delivery system is coated with rapamycin or other suitable drug, agent or compound and delivered intraluminally for the treatment and prevention of neointimal hyperplasia following percutaneous transluminal coronary angiography. The local delivery of the drugs or agents provides for increased effectiveness and lower systemic toxicity.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L21 ANSWER 19 OF 52 USPATFULL

ACCESSION NUMBER: 2002:12284 USPATFULL
TITLE: Arrayed transfection method and uses related thereto
INVENTOR(S): Sabatini, David M., Cambridge, MA, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002006664	A1	20020117
APPLICATION INFO.:	US 2001-817003	A1	20010322 (9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-193580P	20000330 (60)
	US 1999-154737P	19990917 (60)
DOCUMENT TYPE:	Utility	
FILE SEGMENT:	APPLICATION	
LEGAL REPRESENTATIVE:	ROPES & GRAY, ONE INTERNATIONAL PLACE, BOSTON, MA, 02110-2624	
NUMBER OF CLAIMS:	37	
EXEMPLARY CLAIM:	1	
NUMBER OF DRAWINGS:	7 Drawing Page(s)	
LINE COUNT:	2671	

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB An arrayed transfection method of introducing nucleic acid of interest into cells.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L21 ANSWER 20 OF 52 USPATFULL

ACCESSION NUMBER: 2002:10838 USPATFULL
TITLE: Antiproliferative drug and delivery device
INVENTOR(S): Falotico, Robert, Belle Mead, NJ, UNITED STATES
Kopia, Gregory A., Hillsborough, NJ, UNITED STATES
Llanos, Gerard H., Stewartsville, NJ, UNITED STATES
Siekjerka, John, Towaco, NJ, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002005206	A1	20020117
APPLICATION INFO.:	US 2001-850507	A1	20010507 (9)
RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 2000-575480, filed on 19 May 2000, UNKNOWN		
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	APPLICATION		
LEGAL REPRESENTATIVE:	AUDLEY A. CIAMPORCERO JR., JOHNSON & JOHNSON, ONE JOHNSON & JOHNSON PLAZA, NEW BRUNSWICK, NJ, 08933-7003		
NUMBER OF CLAIMS:	15		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	2 Drawing Page(s)		
LINE COUNT:	921		

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB A drug and drug delivery system may be utilized in the treatment of vascular disease. A local delivery system is coated with rapamycin or other suitable drug, agent or compound and delivered intraluminally for the treatment and prevention of neointimal hyperplasia following percutaneous transluminal coronary angiography. The local delivery of the drugs or agents provides for increased effectiveness and lower systemic toxicity.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L21 ANSWER 21 OF 52 USPATFULL

ACCESSION NUMBER: 2002:325826 USPATFULL
TITLE: Mammalian proteins that bind to FKBP12 in a rapamycin-dependent fashion
INVENTOR(S): Sabatini, David M., Baltimore, MD, United States
Erdjument-Bromage, Hediye, New York, NY, United States
Lui, Mary, Kew Gardens, NY, United States
Tempst, Paul, New York, NY, United States
Snyder, Solomon H., Baltimore, MD, United States
PATENT ASSIGNEE(S): The Johns Hopkins University, Baltimore, MD, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6492106	B1	20021210
APPLICATION INFO.:	US 1994-305790		19940914 (8)
RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 1994-265967, filed on 27 Jun 1994		
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	GRANTED		
PRIMARY EXAMINER:	Patterson, Jr., Charles L.		
ASSISTANT EXAMINER:	Kerr, Kathleen		
LEGAL REPRESENTATIVE:	Banner & Witcoff, Ltd.		
NUMBER OF CLAIMS:	12		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	11 Drawing Figure(s); 10 Drawing Page(s)		
LINE COUNT:	2121		

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB A protein complex containing 245 kDa and 35 kDa components, designated RAFT1 and RAFT2 (for Rapamycin And FKBP12 Target) interacts with FKBP12 in a rapamycin-dependent manner. This interaction has the pharmacological characteristics expected from the observed in vivo effects of rapamycin: it occurs at low nanomolar concentrations of rapamycin and is competed by excess FK506. Sequences (330 amino acids total) of tryptic peptides derived from the affinity purified 245 kDa RAFT1 reveals striking homologies to the predicted products of the yeast TOR genes, which were originally identified by mutations that confer rapamycin resistance in yeast. A RAFT1 cDNA was obtained and found to encode a 289 kDa protein (2550 amino acids) that is 43% and 39% identical to TOR2 and TOR1, respectively.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L21 ANSWER 22 OF 52 USPATFULL

ACCESSION NUMBER: 2002:303872 USPATFULL
TITLE: Lipid kinase
INVENTOR(S): Vanhaesebroeck, Bart, London, UNITED KINGDOM
Waterfield, Michael Derek, London, UNITED KINGDOM
PATENT ASSIGNEE(S): Ludwig Institute for Cancer Research, New York, NY,
United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6482623	B1	20021119
	WO 9746688		19971211
APPLICATION INFO.:	US 1998-194640		19981201 (9)
	WO 1997-GB1471		19970530
			19981201 PCT 371 date

	NUMBER	DATE
PRIORITY INFORMATION:	GB 1996-11460	19960601
DOCUMENT TYPE:	Utility	
FILE SEGMENT:	GRANTED	
PRIMARY EXAMINER:	Achutamurthy, Ponnathapu	
ASSISTANT EXAMINER:	Rao, Manjunath N.	
LEGAL REPRESENTATIVE:	Fulbright & Jaworski LLP	
NUMBER OF CLAIMS:	20	
EXEMPLARY CLAIM:	1	
NUMBER OF DRAWINGS:	15 Drawing Figure(s); 18 Drawing Page(s)	
LINE COUNT:	1568	

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The invention relates to a novel lipid kinase which is part of the PI3 Kinase family. PI3 Kinases catalyze the addition of phosphate to inositol generating inositol mono, di and triphosphate. Inositol phosphates have been implicated in regulating intracellular signalling cascades resulting in alternations in gene expression which, amongst other effects, can result in cytoskeletal remodelling and modulation of cellular motility. More particularly the invention relates to a novel human PI3 Kinase, p110.delta. which interacts with p85, has a broad phosphoinositide specificity and is sensitive to the same kinase inhibitors as PI3 Kinase p110.alpha.. However in contrast to previously identified PI3 Kinases which show a ubiquitous pattern of expression, p110.delta. is selectively expressed in leucocytes. Importantly, p110.delta. shows enhanced expression in most melanomas tested and therefore may play a crucial role in regulating the metastatic property exhibited by melanomas. The identification of agents that enhance or reduce p110.delta. activity may therefore prevent cancer metastasis.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L21 ANSWER 23 OF 52 USPATFULL

ACCESSION NUMBER: 2002:291067 USPATFULL
 TITLE: Mammalian proteins that bind to FKBP12 in a rapamycin-dependent fashion
 INVENTOR(S): Sabatini, David M., Baltimore, MD, United States
 Erdjument-Bromage, Hediye, New York, NY, United States
 Lui, Mary, Kew Gardens, NY, United States
 Tempst, Paul, New York, NY, United States
 Snyder, Solomon H., Baltimore, MD, United States
 PATENT ASSIGNEE(S): The Johns Hopkins University, Baltimore, MD, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6476200	B1	20021105
APPLICATION INFO.:	US 1994-265967		19940627 (8)
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	GRANTED		
PRIMARY EXAMINER:	Prouty, Rebecca E.		
ASSISTANT EXAMINER:	Kerr, Kathleen		
LEGAL REPRESENTATIVE:	Banner & Witcoff, Ltd.		
NUMBER OF CLAIMS:	2		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	11 Drawing Figure(s); 10 Drawing Page(s)		
LINE COUNT:	1878		

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB A protein complex containing 245 kDa and 35 kDa components, designated RAFT1 and RAFT2 (for Rapamycin And FKBP12 Target) interacts with FKBP12 in a rapamycin-dependent manner. This interaction has the pharmacological characteristics expected from the observed in vivo effects of rapamycin: it occurs at low nanomolar concentrations of rapamycin and is competed by excess FK506. Sequences (330 amino acids total) of tryptic peptides derived from the affinity purified 245 kDa RAFT1 reveals striking homologies to the predicted products of the yeast TOR genes, which were originally identified by mutations that confer rapamycin resistance in yeast. A RAFT1 cDNA was obtained and found to encode a 289 kDa protein (2550 amino acids) that is 43% and 39% identical to TOR2 and TOR1, respectively.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L21 ANSWER 24 OF 52 USPATFULL

ACCESSION NUMBER: 2002:283146 USPATFULL
 TITLE: Biocatalytic synthesis of galloid organics
 INVENTOR(S): Frost, John W., Okemos, MI, United States
 PATENT ASSIGNEE(S): Board of Trustees operating Michigan State Univerisity, East Lansing, MI, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6472190	B1	20021029
APPLICATION INFO.:	US 2000-527145		20000316 (9)
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	GRANTED		
PRIMARY EXAMINER:	Achutamurthy, Ponnathapu		
ASSISTANT EXAMINER:	Walicka, Malgorzata A.		
LEGAL REPRESENTATIVE:	Harness, Dickey & Pierce, P.L.C.		
NUMBER OF CLAIMS:	22		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	9 Drawing Figure(s); 5 Drawing Page(s)		
LINE COUNT:	1645		

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The present invention provides a bioengineered synthesis scheme for the production of gallic acid from a carbon source. Methods of producing gallic acid from a carbon source based on the synthesis scheme are also

provided. The gallic acid produces from these methods can be further converted to pyrogallol. Methods for the biosynthesis of pyrogallol from gallic acid are also provided.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L21 ANSWER 25 OF 52 USPATFULL

ACCESSION NUMBER: 2002:238643 USPATFULL
TITLE: Antagonists of interleukin-15
INVENTOR(S): Strom, Terry B., Brookline, MA, United States
Maslinski, Wlodzimierz, Warsaw, POLAND
PATENT ASSIGNEE(S): Beth Israel Deaconess Medical Center, Boston, MA,
United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6451308	B1	20020917
APPLICATION INFO.:	US 1999-437585		19991109 (9)
RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 1997-842947, filed on 25 Apr 1997, now patented, Pat. No. US 6001973		

	NUMBER	DATE
PRIORITY INFORMATION:	US 1996-16634P	19960426 (60)
DOCUMENT TYPE:	Utility	
FILE SEGMENT:	GRANTED	
PRIMARY EXAMINER:	Eyler, Yvonne	
ASSISTANT EXAMINER:	Prasad, Sarada	
LEGAL REPRESENTATIVE:	Fish & Richardson P.C.	
NUMBER OF CLAIMS:	11	
EXEMPLARY CLAIM:	1	
NUMBER OF DRAWINGS:	33 Drawing Figure(s); 19 Drawing Page(s)	
LINE COUNT:	2062	

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Disclosed herein are mutant IL-15 polypeptides and methods for using these polypeptides to modulate the immune response in a patient.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L21 ANSWER 26 OF 52 USPATFULL

ACCESSION NUMBER: 2002:202055 USPATFULL
TITLE: Beclin and uses thereof
INVENTOR(S): Levine, Beth C., Briarcliff Manor, NY, United States
PATENT ASSIGNEE(S): The Trustees of Columbia University in the City of New York, New York, NY, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6432914	B1	20020813
APPLICATION INFO.:	US 1999-265630		19990309 (9)
RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 1999-250045, filed on 12 Feb 1999, now abandoned Continuation-in-part of Ser. No. US 1998-40808, filed on 18 Mar 1998, now abandoned Continuation-in-part of Ser. No. WO 1997-US16358, filed on 12 Sep 1997, now abandoned Continuation-in-part of Ser. No. US 1996-712939, filed on 13 Sep 1996, now patented, Pat. No. US 5858669, issued on 12 Jan 1999		
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	GRANTED		
PRIMARY EXAMINER:	Carlson, Karen Cochrane		
ASSISTANT EXAMINER:	Robinson, Hope A.		
LEGAL REPRESENTATIVE:	White, John P., Cooper & Dunham LLP		
NUMBER OF CLAIMS:	3		

EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 82 Drawing Figure(s); 41 Drawing Page(s)
LINE COUNT: 4644
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB This invention provides a method of diagnosing a predisposition to carcinoma in a subject comprising: a) obtaining a nucleic acid sample from the subject; b) determining whether one or more of the subject's beclin alleles or regulatory regions to those alleles are deleted or different from the wild type so as to reduce the subject's expression of polypeptide having tumor suppressor activity; and c) determining whether one or more of the subject's beclin alleles or regulatory regions to those alleles are deleted or changed so as to reduce the subject's ability to mediate autophagy. This invention also provides uses of beclin for treating viral diseases. This invention provides a method for inhibiting viral replication comprising contacting effective amount of Beclin with the virus infected cell, thereby inhibiting the viral replication. This invention also provides a method for inhibiting viral replication comprising contacting induction of the expression of beclin with the virus infected cell, thereby inhibiting the viral replication. This invention also provides a method for treating cancer comprising inducing increased expression of Beclin, thereby restoring autophagy as well as a method for treating cancer which comprises administering to the subject a therapeutically effective amount of beclin so as to restore autophagy.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L21 ANSWER 27 OF 52 USPATFULL

ACCESSION NUMBER: 2002:194721 USPATFULL
TITLE: Immunosuppressive structural definition of IL-10
INVENTOR(S): Bromberg, Jonathan S., New York, NY, United States
Ding, YaoZhong, Forest Hills, NY, United States
Qin, LiHui, New York, NY, United States
PATENT ASSIGNEE(S): The Regents of the University of Michigan, Ann Arbor, MI, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6428985	B1	20020806
APPLICATION INFO.:	US 1999-452624		19991130 (9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 1998-110601P	19981202 (60)
DOCUMENT TYPE:	Utility	
FILE SEGMENT:	GRANTED	
PRIMARY EXAMINER:	Stucker, Jeffrey	
ASSISTANT EXAMINER:	Seharaseyon, Jegatheesan	
LEGAL REPRESENTATIVE:	Fulbright & Jaworski LLP	
NUMBER OF CLAIMS:	21	
EXEMPLARY CLAIM:	1	
NUMBER OF DRAWINGS:	13 Drawing Figure(s); 9 Drawing Page(s)	
LINE COUNT:	3383	

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Disclosed is the surprising discovery that a single amino acid provides the demarcation between the immunosuppressive and immunostimulatory properties of the cytokine, IL-10. The present invention thus provides mammalian and human IL-10 genes and polypeptides that have immunosuppressive properties, without immunostimulatory side-effects. Also provided are various methods of using the new IL-10 constructs, both in vitro and in vivo, particularly in sole or combination therapies involving immunosuppression, such as in the treatment of inflammatory diseases and disorders, and in transplantation.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L21 ANSWER 28 OF 52 USPATFULL

ACCESSION NUMBER: 2001:233124 USPATFULL
TITLE: Inhibition of GSK-3 beta
INVENTOR(S): Hoeflich, Klaus, Toronto, Canada
Woodgett, James, Toronto, Canada
Luo, Juan, Toronto, Canada

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2001053351	A1	20011220
APPLICATION INFO.:	US 2000-747552	A1	20001222 (9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 1999-172064P	19991223 (60)
DOCUMENT TYPE:	Utility	
FILE SEGMENT:	APPLICATION	
LEGAL REPRESENTATIVE:	Pamela J. Sherwood, BOZICEVIC, FIELD & FRANCIS LLP, Suite 200, 200 Middlefield Road, Menlo Park, CA, 94025	
NUMBER OF CLAIMS:	18	
EXEMPLARY CLAIM:	1	
NUMBER OF DRAWINGS:	7 Drawing Page(s)	
LINE COUNT:	1099	

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The activity of NF-.kappa.B is modulated through the effects of GSK-3 on NF-.kappa.B activity. Inhibition or down-regulation of GSK-3 results in decreased NF-.kappa.B activity. Inappropriate activation of NF-.kappa.B has been linked to inflammation and hyperproliferative disorders. Development of modulatory strategies provide a novel therapeutic tool for the treatment or prevention of various diseases. Methods are also provided for enhanced killing of tumor cells through the sensitization action of GSK-3 inhibition, when administered in conjunction with apoptosis inducing ligands of TNFR1. Transgenic animals defective in GSK-3 function are also provided.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L21 ANSWER 29 OF 52 USPATFULL

ACCESSION NUMBER: 2001:206421 USPATFULL
TITLE: Drosophila melanogaster **p70 S6**
kinase
INVENTOR(S): Stewart, Mary, Fargo, ND, United States
Kozma, Sara, Hesingue, France
Thomas, George, Hesingue, France

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2001042254	A1	20011115
	US 6534311	B2	20030318
APPLICATION INFO.:	US 2001-817310	A1	20010326 (9)
RELATED APPLN. INFO.:	Continuation of Ser. No. US 1999-230247, filed on 16 May 1999, ABANDONED A 371 of International Ser. No. WO 1997-EP3680, filed on 11 Jul 1997, UNKNOWN		

	NUMBER	DATE
PRIORITY INFORMATION:	GB 1996-15498	19960724
DOCUMENT TYPE:	Utility	
FILE SEGMENT:	APPLICATION	
LEGAL REPRESENTATIVE:	THOMAS HOXIE, NOVARTIS CORPORATION, PATENT AND TRADEMARK DEPT, 564 MORRIS AVENUE, SUMMIT, NJ, 079011027	

NUMBER OF CLAIMS: 9
EXEMPLARY CLAIM: 1
LINE COUNT: 1746

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The invention provides *Drosophila melanogaster* p70.sup.S6K, as well as nucleic acids encoding this kinase. The sequence of *Drosophila* p70.sup.S6K and the gene encoding it are represented in SEQ ID No. 2 and 1 respectively. The invention moreover provides mutated forms of *Drosophila* p70.sup.S6K, including constitutively active and dominant negative forms thereof, which are useful in the study of p70.sup.S6K activity. Furthermore, the invention provides expression systems which produce *Drosophila* p70.sup.S6K in *Drosophila* and other organisms, and in particular systems in which expression of *Drosophila* p70.sup.S6K has been modulated so as to facilitate the study of its activity.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L21 ANSWER 30 OF 52 USPATFULL

ACCESSION NUMBER: 2001:173359 USPATFULL
TITLE: Human signal transduction serine/threonine kinase
INVENTOR(S): Norris, Tyrrell Errick, New Castle, DE, United States
Moore, William Craig, West Grove, PA, United States
Silberstein, David Shay, Kenneth Square, PA, United States
PATENT ASSIGNEE(S): Zeneca Limited, London, United Kingdom (non-U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6300098	B1	20011009
APPLICATION INFO.:	US 1999-468442		19991221 (9)
RELATED APPLN. INFO.:	Division of Ser. No. US 1999-340993, filed on 25 Jun 1999, now patented, Pat. No. US 6034228 Continuation-in-part of Ser. No. US 1998-211930, filed on 15 Dec 1998, now patented, Pat. No. US 5962265		

	NUMBER	DATE
PRIORITY INFORMATION:	GB 1997-139726851	19971219
DOCUMENT TYPE:	Utility	
FILE SEGMENT:	GRANTED	
PRIMARY EXAMINER:	Schwartzman, Robert A.	
ASSISTANT EXAMINER:	Epps, Janet	
NUMBER OF CLAIMS:	3	
EXEMPLARY CLAIM:	1	
LINE COUNT:	2340	

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB An isolated and purified human Ste20-like serine/threonine signal transduction kinase is described. A cDNA sequence which encodes the native signal transduction molecule is disclosed as well as the structural coding region and the amino acid residue sequence. Methods are provided which employ the sequences to identify compounds that modulate the biological and/or pharmacological activity of the transduction molecule and hence regulate cell physiology. Biologically-effective **antisense** molecules, as well as dominant negative mutant versions of the biomolecule are described which are suitable for therapeutic use. The invention is also drawn toward the diagnosis, prevention, and treatment of pathophysiological disorders mediated by the signal transduction molecule.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L21 ANSWER 31 OF 52 USPATFULL

ACCESSION NUMBER: 2001:117165 USPATFULL

TITLE: Human Ste20-like stress activated serine/threonine kinase

INVENTOR(S): Moore, William Craig, West Grove, PA, United States
Norris, Tyrrell Errick, New Castle, DE, United States
Silberstein, David Shay, Kennett Square, PA, United States

PATENT ASSIGNEE(S): Zeneca Ltd., United Kingdom (non-U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6265560	B1	20010724
APPLICATION INFO.:	US 1998-152406		19980914 (9)

	NUMBER	DATE
PRIORITY INFORMATION:	GB 1997-19920	19970919
DOCUMENT TYPE:	Utility	
FILE SEGMENT:	GRANTED	
PRIMARY EXAMINER:	Schwartzman, Robert A.	
ASSISTANT EXAMINER:	Larson, Thomas G.	
LEGAL REPRESENTATIVE:	Mitchell, Kenneth F.	
NUMBER OF CLAIMS:	1	
EXEMPLARY CLAIM:	1	
NUMBER OF DRAWINGS:	10 Drawing Figure(s); 10 Drawing Page(s)	
LINE COUNT:	2395	

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB A novel human signal-transduction kinase polypeptide is described which is expressed at a particularly high level in human leukocytes. A full length cDNA which encodes the novel stress-activated serine/threonine kinase polypeptide is disclosed as well as the interior structural region and the amino acid residue sequence of the native biological molecule. Methods are provided to identify compounds that modulate the biological activity of the human Ste20-like stress-activated serine/threonine signal transduction kinase.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L21 ANSWER 32 OF 52 USPATFULL

ACCESSION NUMBER: 2001:116764 USPATFULL

TITLE: Ataxia-telangiectasia gene and its genomic organization

INVENTOR(S): Shiloh, Yosef, Tel Aviv, Israel

PATENT ASSIGNEE(S): Ramot-University Authority for Applied Research and Industrial Development, Tel Aviv, Israel (non-U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6265158	B1	20010724
	WO 9636691		19961121
APPLICATION INFO.:	US 1998-952014		19980202 (8)
	WO 1996-US7025		19960516
			19980202 PCT 371 date
			19980202 PCT 102(e) date
RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 1996-629001, filed on 8 Apr 1996, now patented, Pat. No. US 5858661		
	Continuation-in-part of Ser. No. US 1995-441822, filed on 16 May 1995, now patented, Pat. No. US 5756288		
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	GRANTED		
PRIMARY EXAMINER:	Jones, W. Gary		
ASSISTANT EXAMINER:	Goldberg, Jeanine		
LEGAL REPRESENTATIVE:	Kohn & Associates		
NUMBER OF CLAIMS:	7		
EXEMPLARY CLAIM:	1,7		

NUMBER OF DRAWINGS: 4 Drawing Figure(s); 2 Drawing Page(s)

LINE COUNT: 3109

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB A purified and isolated gene, designated ATM, mutations of which cause ataxia-telangiectasia, its genomic organization, methods for the detection of the defective gene, the purified polypeptide encoded by the defective gene, and antibodies recognizing the defective protein.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L21 ANSWER 33 OF 52 USPATFULL

ACCESSION NUMBER: 2001:107664 USPATFULL

TITLE: Stimulus-inducible protein kinase complex and methods of use therefor

INVENTOR(S): Mercurio, Frank, San Diego, CA, United States

Zhu, Hengyi, San Diego, CA, United States

Barbosa, Miguel, San Diego, CA, United States

Li, Jian Wu, San Diego, CA, United States

Murray, Brion W., San Diego, CA, United States

PATENT ASSIGNEE(S): Signal Pharmaceuticals, Inc., San Diego, CA, United States (U.S. corporation)

NUMBER	KIND	DATE
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PATENT INFORMATION:	US 6258579	B1 20010710
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APPLICATION INFO.:	US 1997-910820	19970813 (8)
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RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 1996-697393, filed on 26 Aug 1996, now patented, Pat. No. US 5972674	
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DOCUMENT TYPE:	Utility	
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FILE SEGMENT:	GRANTED	
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PRIMARY EXAMINER:	Patterson, Jr., Charles L.	
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LEGAL REPRESENTATIVE:	Mathews, Collins, Shepherd & Gould, P.A.	
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NUMBER OF CLAIMS:	4	
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EXEMPLARY CLAIM:	1	
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NUMBER OF DRAWINGS:	30 Drawing Figure(s); 28 Drawing Page(s)	
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LINE COUNT:	1713	
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CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Compositions and methods are provided for treating NF-.kappa.B-related conditions. In particular, the invention provides a stimulus-inducible IKK signalsome, and components and variants thereof. An IKK signalsome or component thereof may be used, for example, to identify antibodies and other modulating agents that inhibit or activate signal transduction via the NF-.kappa.B cascade. IKK signalsome, components thereof and/or modulating agents may also be used for the treatment of diseases associated with NF-.kappa.B activation.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L21 ANSWER 34 OF 52 USPATFULL

ACCESSION NUMBER: 2001:48208 USPATFULL

TITLE: Ataxia-telangiectasia gene

INVENTOR(S): Shiloh, Yosef, Tel Aviv, Israel

Tagle, Danilo A., Gaithersburg, MD, United States

Collins, Francis, Rockville, MD, United States

PATENT ASSIGNEE(S): The United States of America as represented by the Department of Health and Human Services, Washington, DC, United States (U.S. government)
Ramot University Authority for Applied Research and Industrial Dev., Israel (non-U.S. corporation)

NUMBER	KIND	DATE
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PATENT INFORMATION:	US 6211336	B1 20010403
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	WO 9636695	19961121
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APPLICATION INFO.: US 1998-952127 19980226 (8)
WO 1996-US7040 19960516
19980226 PCT 371 date
19980226 PCT 102(e) date
RELATED APPLN. INFO.: Continuation-in-part of Ser: No. US 1995-508836, filed
on 28 Jul 1995, now patented, Pat. No. US 5777093
Continuation-in-part of Ser. No. US 1995-493092, filed
on 21 Jun 1995, now patented, Pat. No. US 5728807
Continuation-in-part of Ser. No. US 1995-441822, filed
on 16 May 1995, now patented, Pat. No. US 5756288
DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Cintins, Marianne M.
ASSISTANT EXAMINER: Delacroix-Muirheid, C.
LEGAL REPRESENTATIVE: Kohn & Associates
NUMBER OF CLAIMS: 5
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 10 Drawing Figure(s); 4 Drawing Page(s)
LINE COUNT: 2279

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB There is provided a purified amino acid sequence selected from the group
of Sequence ID No.: 3 and analogs thereof and mutations of Sequence ID
No.: 3 which cause ataxia-telangiectasia. Also provided is a purified
amino acid sequence as set forth in Sequence ID No.: 3 and analogs
thereof.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L21 ANSWER 35 OF 52 USPATFULL

ACCESSION NUMBER: 2001:36598 USPATFULL
TITLE: Mutated forms of the ataxia-telangiectasia gene and
method to screen for a partial A-T phenotype
INVENTOR(S): Shiloh, Yosef, Tel Aviv, Israel
PATENT ASSIGNEE(S): Ramot-University Authority for Applied Research and
Industrial Development Ltd., Tel Aviv, Israel (non-U.S.
corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6200749	B1	20010313
APPLICATION INFO.:	US 1996-642274		19960503 (8)
RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 1996-629001, filed on 8 Apr 1996 Continuation-in-part of Ser. No. US 1995-441822, filed on 16 May 1995, now patented, Pat. No. US 5756288		
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	Granted		
PRIMARY EXAMINER:	Arthur, Lisa B.		
LEGAL REPRESENTATIVE:	Kohn & Associates		
NUMBER OF CLAIMS:	5		
EXEMPLARY CLAIM:	1,4		
NUMBER OF DRAWINGS:	8 Drawing Figure(s); 2 Drawing Page(s)		
LINE COUNT:	3090		

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB A purified and isolated gene, designated ATM, is described mutations of
which cause ataxia-telangiectasia and its genomic organization.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L21 ANSWER 36 OF 52 MEDLINE DUPLICATE 2
ACCESSION NUMBER: 2001408068 MEDLINE
DOCUMENT NUMBER: 21352732 PubMed ID: 11459796
TITLE: Characterization of signal transduction pathway in
neurotropic action of angiotensin II in brain neurons.

AUTHOR: Yang H; Wang X; Raizada M K
 CORPORATE SOURCE: Department of Physiology, College of Medicine, and McKnight Brain Institute, University of Florida, Gainesville, Florida 32610, USA.
 CONTRACT NUMBER: HL-33610 (NHLBI)
 SOURCE: ENDOCRINOLOGY, (2001 Aug) 142 (8) 3502-11.
 Journal code: 0375040. ISSN: 0013-7227.
 PUB. COUNTRY: United States
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
 LANGUAGE: English
 FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
 ENTRY MONTH: 200108
 ENTRY DATE: Entered STN: 20010820
 Last Updated on STN: 20010820
 Entered Medline: 20010816

AB Interaction of angiotensin II with the neuronal angiotensin type 1 receptor stimulates the PI3K signaling pathway. Our objective in this study was to investigate the hypothesis that the PI3K cascade regulates the neurotropic actions of angiotensin II in rat brain neurons. We followed growth associated protein-43 expression and neurite extension as markers of neurotropic activity. Angiotensin II, through its interaction with the angiotensin type 1 receptor, increased growth associated protein-43 expression and neurite extension. These effects were abolished by pretreatment of neurons with wortmannin and rapamycin, but not by PD 98059. **Antisense** oligonucleotides specific for **p70(S6) kinase** also inhibited angiotensin II-stimulated neurotropic activity. These data confirm the involvement of PI3K and **p70(S6) kinase** in angiotensin II-mediated neurotropic action. Further support for this was provided by the observation that angiotensin II caused a time-dependent stimulation of **p70(S6) kinase** by an angiotensin type 1 receptor-mediated process. We also found that the neurotropic actions of angiotensin II are mediated by plasminogen activator inhibitor-1. Evidence for this includes 1) angiotensin II-stimulated neuronal plasminogen activator inhibitor-1 gene expression, 2) potent neurotropic action of exogenous plasminogen activator inhibitor-1, and 3) inhibitory neurotropic effect of angiotensin II by **antisense** oligonucleotide-mediated depletion of plasminogen activator inhibitor-1. Finally, we found that the neurotropic action of plasminogen activator inhibitor-1 is not blocked by either angiotensin type 1 receptor antagonist or inhibitors of PI3K or **p70(S6) kinase**, indicating that the plasminogen activator inhibitor-1 step is downstream from the **p70(S6) kinase**. These observations demonstrate that angiotensin II is a neurotropic hormone that engages a distinct PI3K-**p70(S6) kinase**-plasminogen activator inhibitor-1 signaling pathway for this action.

L21 ANSWER 37 OF 52 USPATFULL

ACCESSION NUMBER: 2000:128305 USPATFULL
 TITLE: **Antisense** modulation of PDK-1 expression
 INVENTOR(S): Monia, Brett P., La Costa, CA, United States
 Cowser, Lex M., Carlsbad, CA, United States
 PATENT ASSIGNEE(S): Isis Pharmaceutical Inc., Carlsbad, CA, United States
 (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6124272		20000926
APPLICATION INFO.:	US 1999-289466		19990409 (9)
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	Granted		
PRIMARY EXAMINER:	Elliott, George C.		
ASSISTANT EXAMINER:	Epps, Janet		

LEGAL REPRESENTATIVE: Law Offices of Jane Massey Licata
NUMBER OF CLAIMS: 21
EXEMPLARY CLAIM: 1
LINE COUNT: 3074

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB **Antisense** compounds, compositions and methods are provided for modulating the expression of PDK-1. The compositions comprise **antisense** compounds, particularly **antisense** oligonucleotides, targeted to nucleic acids encoding PDK-1. Methods of using these compounds for modulation of PDK-1 expression and for treatment of diseases associated with expression of PDK-1 are provided.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L21 ANSWER 38 OF 52 USPATFULL

ACCESSION NUMBER: 2000:28123 USPATFULL
TITLE: Human signal transduction serine/threonine kinase
INVENTOR(S): Norris, Tyrrell Errick, New Castle, DE, United States
Moore, William Craig, West Grove, PA, United States
Silberstein, David Shay, Kennett Square, PA, United States
PATENT ASSIGNEE(S): Zeneca Limited, London, United Kingdom (non-U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6034228		20000307
APPLICATION INFO.:	US 1999-340993		19990625 (9)
RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 1998-211930, filed on 15 Dec 1998		
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	Granted		
PRIMARY EXAMINER:	Elliott, George C.		
ASSISTANT EXAMINER:	Epps, Janet		
LEGAL REPRESENTATIVE:	Higgins, Esq., Patrick H.		
NUMBER OF CLAIMS:	8		
EXEMPLARY CLAIM:	1		
LINE COUNT:	2808		

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB An isolated and purified human Ste20-like serine/threonine signal transduction kinase is described. A cDNA sequence which encodes the native signal transduction molecule is disclosed as well as the structural coding region and the amino acid residue sequence. Methods are provided which employ the sequences to identify compounds that modulate the biological and/or pharmacological activity of the transduction molecule and hence regulate cell physiology. Biologically-effective **antisense** molecules, as well as dominant negative mutant versions of the biomolecule are described which are suitable for therapeutic use. The invention is also drawn toward the diagnosis, prevention, and treatment of pathophysiological disorders mediated by the signal transduction molecule.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L21 ANSWER 39 OF 52 MEDLINE

DUPLICATE 3

ACCESSION NUMBER: 2001383027 MEDLINE
DOCUMENT NUMBER: 21062689 PubMed ID: 11102805
TITLE: Inhibition of PDK-1 activity causes a reduction in cell proliferation and survival.
AUTHOR: Flynn P; Wongdaggar M; Zavar M; Dean N M; Stokoe D
CORPORATE SOURCE: Cancer Research Institute, University of California, San Francisco, California 94115, USA.
CONTRACT NUMBER: .R01 CA79548 (NCI)
SOURCE: CURRENT BIOLOGY, (2000 Nov 16) 10 (22) 1439-42.

Journal code: 9107782. ISSN: 0960-9822.

PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200107
ENTRY DATE: Entered STN: 20010709
Last Updated on STN: 20020420
Entered Medline: 20010705

AB 3-Phosphoinositide-dependent kinase-1 (PDK-1) was identified by its ability to phosphorylate and activate protein kinase B (PKB) in vitro [1,2] and can phosphorylate and activate additional protein kinases in the AGC family in vitro [3-6]. Its role in vivo has, however, only begun to be addressed. We used **antisense** oligonucleotides directed against PDK-1 expression to explore the role of PDK-1 in human glioblastoma cells (U87-MG), which express a mutant PTEN allele. Reduction in PDK-1 levels resulted in inhibition of PKB activity, and a reduction in phosphorylation on Thr308 and Ser473 of PKB. **p70 S6 kinase** (p70(S6K)) activity was also reduced. Cell proliferation was dramatically inhibited following treatment with PDK-1 **antisense** oligonucleotides, due to a combination of decreased cell doubling and an increase in apoptosis. This is in contrast to direct inhibition of phosphoinositide 3-OH kinase (PI 3-kinase), which results in G1 arrest with no effect on apoptosis. This study confirms both PKB and p70(S6K) as in vivo substrates for PDK-1. The effect of acute PDK-1 loss on cell proliferation and survival suggests the involvement of PI 3-kinase dependent and independent signaling events, and implicates PDK-1 as a potential therapeutic target for human neoplasms.

L21 ANSWER 40 OF 52 MEDLINE DUPLICATE 4

ACCESSION NUMBER: 2000441834 MEDLINE
DOCUMENT NUMBER: 20403862 PubMed ID: 10944430
TITLE: Differential regulation of phospholipase C-beta isozymes in cardiomyocyte hypertrophy.
AUTHOR: Schnabel P; Mies F; Nohr T; Geisler M; Bohm M
CORPORATE SOURCE: Klinik III fur Innere Medizin, Universitat zu Koln, Joseph-Stelzmann-Strasse 9, Cologne, 50924, Germany.
SOURCE: BIOCHEMICAL AND BIOPHYSICAL RESEARCH COMMUNICATIONS, (2000 Aug 18) 275 (1) 1-6.
Journal code: 0372516. ISSN: 0006-291X.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200009
ENTRY DATE: Entered STN: 20000928
Last Updated on STN: 20000928
Entered Medline: 20000921

AB Cardiac hypertrophy is a major predictor of heart failure and of morbidity and mortality in developed countries. Many hormones and growth factors induce cardiac hypertrophy via activation of members of the phospholipase C (PLC) family. The expression pattern of the PLCbeta isozyme subfamily was investigated in neonatal rat cardiomyocytes after stimulation with different hypertrophic stimuli. Under control conditions and after stimulation with norepinephrine, cardiomyocytes expressed similar amounts of PLCbeta3 mRNA. In the presence of fetal calf serum (FCS), additional expression of PLCbeta1 was induced. Growth hormone (GH) and insulin-like growth factor-I (IGF-I) both induced a substantial increase in PLCbeta3 mRNA expression. The response to GH could not be abolished by the IGF-I receptor blocker IGF-I analogue indicating an IGF-I-independent action of GH. The upregulation of PLCbeta3 by IGF-I was abolished by preincubation of cardiomyocytes with the IGF-I receptor antagonist IGF-I analogue, the tyrosine kinase inhibitor genistein, the extracellular signal-related kinase (ERK) inhibitor PD 98059, the phosphatidylinositol-3- (PI-3) kinase

inhibitor wortmannin and the **p70 S6 kinase** inhibitor rapamycin. Induction of the immediate early genes c-myc, c-fos, and c-jun by IGF-I was abolished by preincubation with **antisense** oligos against PLCbeta3. It is concluded that the expression of PLCbeta isozymes in cardiomyocytes is differentially regulated by different hypertrophic stimuli. The upregulation of PLCbeta3 by IGF-I is dependent on the activity of tyrosine kinase, ERK, PI3 kinase, and **p70 S6 kinase** and PLCbeta3 expression seems to be required for the induction of immediate early genes by IGF-I. The involvement of the PLCbeta subfamily in signal transduction of receptors other than G-protein-coupled receptors is suggested.
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L21 ANSWER 41 OF 52 USPATFULL

ACCESSION NUMBER: 1999:163828 USPATFULL
TITLE: Antagonists of interleukin-15
INVENTOR(S): Strom, Terry B., Brookline, MA, United States
Maslinski, Wlodzimierz, Warsaw, Poland
PATENT ASSIGNEE(S): Beth Israel Deaconess Medical Center, Boston, MA,
United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6001973		19991214
APPLICATION INFO.:	US 1997-842947		19970425 (8)
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	Granted		
PRIMARY EXAMINER:	Mertz, Prema		
LEGAL REPRESENTATIVE:	Fish & Richardson, P.C.		
NUMBER OF CLAIMS:	9		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	21 Drawing Figure(s); 18 Drawing Page(s)		
LINE COUNT:	1798		

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Disclosed herein are mutant IL-15 polypeptides and methods for using these polypeptides to modulate the immune response in a patient.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L21 ANSWER 42 OF 52 USPATFULL

ACCESSION NUMBER: 1999:146335 USPATFULL
TITLE: Nucleic acids encoding novel human serine/threonine protein kinases
INVENTOR(S): Bandman, Olga, Mountain View, CA, United States
Goli, Surya K., Sunnyvale, CA, United States
Hillman, Jennifer L., San Jose, CA, United States
PATENT ASSIGNEE(S): Incyte Pharmaceuticals, Inc., Palo Alto, CA, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5985635		19991116
APPLICATION INFO.:	US 1996-749902		19961115 (8)
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	Granted		
PRIMARY EXAMINER:	Teng, Sally P.		
LEGAL REPRESENTATIVE:	Incyte Pharmaceuticals, Inc.		
NUMBER OF CLAIMS:	6		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	12 Drawing Figure(s); 12 Drawing Page(s)		
LINE COUNT:	2520		

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The present invention provides human serine/threonine kinase (HSTK) and polynucleotides which identify and encode HSTK. The invention also

provides genetically engineered expression vectors and host cells comprising the nucleic acid sequences encoding HSTK and a method for producing HSTK. The invention also provides for use of HSTK and agonists, antibodies, or antagonists specifically binding HSTK, in the prevention and treatment of diseases associated with expression of HSTK. Additionally, the invention provides for the use of **antisense** molecules to polynucleotides encoding HSTK for the treatment of diseases associated with the expression of HSTK. The invention also provides diagnostic assays which utilize the polynucleotide, or fragments or the complement thereof, and antibodies specifically binding HSTK.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L21 ANSWER 43 OF 52 USPATFULL

ACCESSION NUMBER: 1999:132563 USPATFULL
 TITLE: Stimulus-inducible protein kinase complex and methods of use therefor
 INVENTOR(S): Mercurio, Frank, San Diego, CA, United States
 Zhu, Hengyi, San Diego, CA, United States
 Barbosa, Miguel, San Diego, CA, United States
 PATENT ASSIGNEE(S): Signal Pharmaceuticals, Inc., San Diego, CA, United States (U.S. corporation)

	NUMBER	KIND	DATE
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PATENT INFORMATION:	US 5972674		19991026
APPLICATION INFO.:	US 1996-697393		19960826 (8)
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	Granted		
PRIMARY EXAMINER:	Chan, Christina Y.		
ASSISTANT EXAMINER:	Nolan, Patrick J.		
LEGAL REPRESENTATIVE:	SEED and BERRY LLP		
NUMBER OF CLAIMS:	2		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	11 Drawing Figure(s); 6 Drawing Page(s)		
LINE COUNT:	945		

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Compositions and methods are provided for treating NF-.kappa.B-related conditions. In particular, the invention provides a stimulus-inducible I.kappa.B.alpha. kinase complex, and components and variants thereof. I.kappa.B.alpha. kinase complex may be used, for example, to identify antibodies and other agents that inhibit or activate signal transduction via the NF-.kappa.B cascade. I.kappa.B.alpha. kinase complex, components thereof and/or such agents may also be used for the treatment of diseases associated with NF-.kappa.B activation.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L21 ANSWER 44 OF 52 USPATFULL

ACCESSION NUMBER: 1999:121168 USPATFULL
 TITLE: Human signal transduction serine/threonine kinase
 INVENTOR(S): Norris, Tyrrell Errick, New Castle, DE, United States
 Moore, William Craig, West Grove, PA, United States
 Silberstein, David Shay, Kennett Square, PA, United States
 PATENT ASSIGNEE(S): Zeneca Limited, London, United Kingdom (non-U.S. corporation)

	NUMBER	KIND	DATE
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PATENT INFORMATION:	US 5962265		19991005
APPLICATION INFO.:	US 1998-211930		19981215 (9)

NUMBER	DATE
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PRIORITY INFORMATION: GB 1997-26851 19971219
DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Degen, Nancy
ASSISTANT EXAMINER: Epps, Janet
LEGAL REPRESENTATIVE: Higgins, Patrick H.
NUMBER OF CLAIMS: 9
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 1 Drawing Figure(s); 18 Drawing Page(s)
LINE COUNT: 3120

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB A novel human signal-transduction kinase polypeptide is described which is expressed at a particularly high level in tissues of the human immune system. A full length cDNA which encodes the novel signal transduction serine/threonine kinase polypeptide is disclosed as well as the interior structural region and the amino acid residue sequence of the native biological molecule. Methods are provided to identify compounds that modulate the biological activity of the human Ste20-like serine/threonine signal transduction kinase.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L21 ANSWER 45 OF 52 USPATFULL

ACCESSION NUMBER: 1999:4329 USPATFULL
TITLE: Ataxia-telangiectasia gene and its genomic organization
INVENTOR(S): Shiloh, Yosef, Tel Aviv, Israel
PATENT ASSIGNEE(S): RAMOT-University Authority for Applied Research and Industrial Development, Tel Aviv, Israel (non-U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5858661		19990112
APPLICATION INFO.:	US 1996-629001		19960408 (8)
RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 1995-441822, filed on 16 May 1995, now patented, Pat. No. US 5756288		
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	Granted		
PRIMARY EXAMINER:	Arthur, Lisa B.		
LEGAL REPRESENTATIVE:	Kohn & Associates		
NUMBER OF CLAIMS:	9		
EXEMPLARY CLAIM:	1,7		
NUMBER OF DRAWINGS:	6 Drawing Figure(s); 2 Drawing Page(s)		
LINE COUNT:	3461		

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB A purified and isolated gene, designated ATM, mutations of which cause ataxia-telangiectasia and its genomic organization is disclosed. Methods and a kit for the detection of carriers of mutations of the ATM gene are provided by analysis of nucleic acids isolated from patients including in situ hybridization, Northern blotting and reverse transcriptase--polymerase chain reaction, Southern blotting, single strand conformational polymorphism, restriction endonuclease fingerprinting (REF), PCR amplification and DNA-chip analysis.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L21 ANSWER 46 OF 52 MEDLINE

DUPLICATE 5

ACCESSION NUMBER: 1999185088 MEDLINE
DOCUMENT NUMBER: 99185088 PubMed ID: 10085104
TITLE: Growth hormone-dependent differentiation of 3T3-F442A preadipocytes requires Janus kinase/signal transducer and activator of transcription but not mitogen-activated protein kinase or p70 S6 kinase

signaling.

AUTHOR: Yarwood S J; Sale E M; Sale G J; Houslay M D; Kilgour E; Anderson N G

CORPORATE SOURCE: Division of Biochemistry and Molecular Biology, Institute of Biological and Life Sciences, University of Glasgow, Glasgow G12 8QQ, Scotland, United Kingdom.

SOURCE: JOURNAL OF BIOLOGICAL CHEMISTRY, (1999 Mar 26) 274 (13) 8662-8.
Journal code: 2985121R. ISSN: 0021-9258.

PUB. COUNTRY: United States

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 199904

ENTRY DATE: Entered STN: 19990511
Last Updated on STN: 20000303
Entered Medline: 19990429

AB The signals mediating growth hormone (GH)-dependent differentiation of 3T3-F442A preadipocytes under serum-free conditions have been studied. GH priming of cells was required before the induction of terminal differentiation by a combination of epidermal growth factor, tri-iodothyronine, and insulin. Cellular depletion of Janus kinase-2 (JAK-2) using **antisense** oligodeoxynucleotides (ODNs) prevented GH-stimulated JAK-2 and signal transducer and activator of transcription (STAT)-5 tyrosine phosphorylation and severely attenuated the ability of GH to promote differentiation. Although p42(MAPK)/p44(MAPK) mitogen-activated protein kinases were activated during GH priming, treatment of cells with PD 098059, which prevented activation of these kinases, did not block GH priming. However, **antisense** ODN-mediated depletion of mitogen-activated protein kinases from the cells showed that their expression was necessary for terminal differentiation. Similarly, although p70(s6k) was activated during GH priming, pretreatment of cells with rapamycin, which prevented the activation of p70(s6k), had no effect on GH priming. However, rapamycin did partially block epidermal growth factor, tri-iodothyronine, and insulin-stimulated terminal differentiation. By contrast, cellular depletion of STAT-5 with **antisense** ODNs completely abolished the ability of GH to promote differentiation. These results indicate that JAK-2, acting specifically via STAT-5, is necessary for GH-dependent differentiation of 3T3-F442A preadipocytes. Activation of p42(MAPK)/p44(MAPK) and p70(s6k) is not essential for the promotion of differentiation by GH, although these signals are required for GH-independent terminal differentiation.

L21 ANSWER 47 OF 52 CAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER: 1998:206541 CAPLUS

DOCUMENT NUMBER: 128:317590

TITLE: Up-regulation of insulin-like growth factor binding protein-5 is independent of muscle cell differentiation, sensitive to rapamycin, but insensitive to wortmannin and LY294002

AUTHOR(S): Rousse, Sophie; Montarras, Didier; Pinset, Christian; Dubois, Catherine

CORPORATE SOURCE: Institut National de la Sante et de la Recherche Medicale, U. 142, Hopital Saint Antoine, Paris, 75571, Fr.

SOURCE: Endocrinology (1998), 139(4), 1487-1493
CODEN: ENDOAO; ISSN: 0013-7227

PUBLISHER: Endocrine Society

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Skeletal myoblast differentiation is stimulated by insulin-like growth factors (IGFs). The autocrine action of IGFs is mediated through the type-1 IGF receptor (IGFR-1) and modulated by IGF binding proteins (IGFBPs) secreted by the cells. The mouse C2 myoblast cell line stably

transfected with a vector producing IGF-II **antisense** RNA was used to show that specific IGFBP expression changes with the state of the cells: high levels of IGFBP-2 mRNA were found only in proliferating myoblasts, whereas IGFBP-3 mRNA was induced in quiescent cells. Secretion of IGFBP5 was strongly stimulated during differentiation. Insulin and IGF dose-response expts. showed that up-regulation of IGFBP-5 resulted from IGFR-1 activation. Drugs interfering with IGFR-1 signaling and inhibiting myoblast differentiation had different effects on IGFBP-5 up-regulation. Two phosphatidylinositol 3-kinase (PI 3-kinase) inhibitors, wortmannin and LY294002 [2-(4-morpholinyl)-8-phenyl-4H-1-benzopyran-4-one], failed to alter IGFBP-5 up-regulation, which persisted in the absence of differentiation. Rapamycin which indirectly prevents activation of the p70 ribosomal protein-S6 kinase (p70S6k), suppressed IGFBP-5 induction. Because the PI3-kinase inhibitors block p70S6k, neither kinase would be required for IGFR-1-dependent IGFBP-5 induction. In C2 anti-IGF-II myoblasts, IGFBP-5 induction is therefore rapamycin-sensitive and independent of differentiation.

REFERENCE COUNT: 57 THERE ARE 57 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L21 ANSWER 48 OF 52 MEDLINE DUPLICATE 6
 ACCESSION NUMBER: 1998102437 MEDLINE
 DOCUMENT NUMBER: 98102437 PubMed ID: 9427710
 TITLE: The upregulation of p27Kip1 by rapamycin results in G1 arrest in exponentially growing T-cell lines.
 AUTHOR: Kawamata S; Sakaida H; Hori T; Maeda M; Uchiyama T
 CORPORATE SOURCE: Institute for Virus Research, Kyoto University, Japan.
 SOURCE: BLOOD, (1998 Jan 15) 91 (2) 561-9.
 Journal code: 7603509. ISSN: 0006-4971.
 PUB. COUNTRY: United States
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
 LANGUAGE: English
 FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
 ENTRY MONTH: 199802
 ENTRY DATE: Entered STN: 19980224
 Last Updated on STN: 19990129
 Entered Medline: 19980211

AB An immunosuppressant Rapamycin (Rap) has been reported to cause G1 arrest by inhibiting **p70 S6 kinase** and G1 cyclin/cdks kinase activities when added to quiescent cells with mitogens. However, antiproliferative effects of Rap on exponentially growing cells have been poorly investigated. We examined the intracellular events after the treatment of Rap in exponentially growing T cells and found that Rap upregulated a cdks inhibitor, p27Kip1 at both mRNA and protein levels in Rap-sensitive cells. Antiproliferative effect of Rap was mainly ascribed to the inhibition of cyclin E/cdk2 kinase activity through the formation of cyclin E/cdk2-p27Kip1 complex rather than inhibition of **p70 S6 kinase** activity. Furthermore, we showed that Rap-sensitive cells with elevated p27Kip1 expression lost sensitivity to Rap when **antisense** p27Kip1 was introduced, which indicates that the basal level of p27Kip1 is one of the limiting factors that determine the sensitivity to Rap in already cycling cells. These data suggest the presence of a putative threshold level of p27Kip1 at late G1 phase in already cycling cells. Rap may cause G1 arrest by upregulating the amount of p27Kip1 beyond the threshold in some Rap-sensitive cells that are exponentially growing.

L21 ANSWER 49 OF 52 USPATFULL
 ACCESSION NUMBER: 97:78582 USPATFULL
 TITLE: Human signal transduction MAPK kinase
 INVENTOR(S): Seger, Rony, Yavne, Israel
 Seger, Dalia, Yavne, Israel
 Ahn, Natalie G., Boulder, CO, United States
 Krebs, Edwin G., Seattle, WA, United States

PATENT ASSIGNEE(S): The Board of Regents of the University of Washington,
Seattle, WA, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5663314		19970902
APPLICATION INFO.:	US 1995-423399		19950418 (8)
RELATED APPLN. INFO.:	Continuation of Ser. No. US 1992-980608, filed on 20 Nov 1992, now abandoned		
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	Granted		
PRIMARY EXAMINER:	Jagannathan, Vasu S.		
ASSISTANT EXAMINER:	Lathrop, Brian		
LEGAL REPRESENTATIVE:	Christensen O'Connor Johnson & Kindness PLLC		
NUMBER OF CLAIMS:	4		
EXEMPLARY CLAIM:	3		
LINE COUNT:	2297		

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB An isolated nucleic acid molecule which hybridizes under stringent conditions with the nucleic acid shown in SEQ ID NO:32 or its complement or the nucleic acid shown in SEQ ID NO:34 or its complement, and which encodes mitogen activated protein kinase kinase protein.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L21 ANSWER 50 OF 52 CAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER: 1997:166264 CAPLUS

DOCUMENT NUMBER: 126:247009

TITLE: Heregulin-stimulated acetylcholine receptor gene expression in muscle: requirement for MAP kinase and evidence for a parallel inhibitory pathway independent of electrical activity

AUTHOR(S): Altioek, Nedret; Altioek, Soner; Changeux, Jean-Pierre
CORPORATE SOURCE: Neurobiologie Moleculaire, Institut Pasteur, Paris, Fr.

SOURCE: EMBO Journal (1997), 16(4), 717-725

CODEN: EMJODG; ISSN: 0261-4189

PUBLISHER: Oxford University Press

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Binding of heregulin (HRG) to its receptor, ErbB3, results in a dimerization with ErbB2/neu and activation of their intrinsic tyrosine kinases, initiating a cascade of events resulting in the stimulation of acetylcholine receptor (AChR) genes in muscle. Here we have examd. the signaling downstream of the HRG receptor. We show that phosphatidylinositol 3'-kinase (PI3K) and SHC bind to the HRG-activated ErbB3 in myotubes. Subsequently, p70S6 kinase (p70S6k), and MAP kinase ERK2 and thereby p90rsk are activated. However, inhibition of PI3K and p70S6k by wortmannin and rapamycin, resp., failed to antagonize AChR .alpha.-subunit gene expression stimulated by HRG, despite the fact that the activities of the kinases were inhibited. In contrast, these inhibitors elevated AChR .alpha.-subunit mRNA levels, by themselves, independently of muscle elec. activity. On the other hand, the 17-mer **antisense** oligonucleotide, EAS1, caused a specific depletion of ERK2 and eliminated the ability of HRG to stimulate AChR .alpha.-subunit gene expression. These results indicate that HRG stimulates expression of AChR genes via ERK2 activation, and provide a physiol. example of neurotrophic factor-assocd. repression of AChR genes by stimulation of p70S6k activity which may contribute to the expression of adult type AChR genes at the neuromuscular junction.

L21 ANSWER 51 OF 52 USPATFULL

ACCESSION NUMBER: 96:19104 USPATFULL

TITLE: Inhibition of insulin-induced adiposis

INVENTOR(S): Alexander-Bridges, Maria C., Newtonville, MA, United States
 Zhao, Hui-Fen, Brookline, MA, United States
 PATENT ASSIGNEE(S): The General Hospital Corporation, Boston, MA, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5496831		19960305
APPLICATION INFO.:	US 1994-242409		19940513 (8)
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	Granted		
PRIMARY EXAMINER:	Killos, Paul J.		
LEGAL REPRESENTATIVE:	Fish & Richardson		
NUMBER OF CLAIMS:	18		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	15 Drawing Figure(s); 7 Drawing Page(s)		
LINE COUNT:	1269		

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The invention features methods of treating insulin-induced obesity, weight gain, and other conditions associated with hyperinsulinemia.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L21 ANSWER 52 OF 52 MEDLINE DUPLICATE 7
 ACCESSION NUMBER: 95010779 MEDLINE
 DOCUMENT NUMBER: 95010779 PubMed ID: 7926037
 TITLE: Serum alleviates the requirement of the granulocyte-macrophage colony-stimulating factor (GM-CSF)-induced Ras activation for proliferation of BaF3 cells.
 AUTHOR: Sakamaki K; Yonehara S
 CORPORATE SOURCE: Pharmaceutical Basic Research Laboratory, JT Inc., Yokohama, Japan.
 SOURCE: FEBS LETTERS, (1994 Oct 17) 353 (2) 133-7.
 Journal code: 0155157. ISSN: 0014-5793.
 PUB. COUNTRY: Netherlands
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
 LANGUAGE: English
 FILE SEGMENT: Priority Journals
 ENTRY MONTH: 199411
 ENTRY DATE: Entered STN: 19941222
 Last Updated on STN: 20000303
 Entered Medline: 19941123

AB Deletion analysis of the beta subunit of the human granulocyte-macrophage colony-stimulating factor (GM-CSF) receptor previously defined two cytoplasmic regions required for distinct signaling. The membrane-proximal region is responsible for induction of c-myc and pim-1, and is indispensable for GM-CSF-dependent proliferation of mouse BaF3 transfectants. The distal region is required for activation of Ras, Raf-1, MAP kinase and p70 S6 kinase as well as induction of c-fos and c-jun, but is dispensable for GM-CSF-dependent proliferation of transfectants under normal culture conditions containing serum. Here we show that signals induced by the distal region of the beta subunit are also required for proliferation. GM-CSF supported proliferation of BaF3 transfectants expressing the normal beta subunit, even in serum-free medium. However, in the absence of seru, GM-CSF did not support proliferation of BaF3 transfectants that have the beta deletion mutants lacking the distal region. Serum-induced activation of Ras, phosphorylation of MAP kinase and expression of c-fos in parental BaF3 cells and antisense oligonucleotide against c-raf blocked DNA synthesis of BaF3 cells. These results indicate that proliferation of BaF3 cells requires signals induced by the proximal as well as the distal region of the beta subunit of the GM-CSF receptor, and that serum

alleviates the requirement of signals induced by the distal region.

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ribosomal(s)s6(s)kiase) or pp70s6k
L1 2981 (P70(S) S6(S) KINASE) OR SK6 OR (P70(W) P85(S) S6(S) KINASE) OR
(P70(W) P85(S) RIBOSOMAL(S) S6(S) KIASE) OR PP70S6K

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L3 24 DUP REM L2 (0 DUPLICATES REMOVED)

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L3 ANSWER 1 OF 24 USPATFULL

ACCESSION NUMBER: 2003:114491 USPATFULL
TITLE: Assay for measuring enzyme activity in vivo
INVENTOR(S): Craig, Roger Kingdon, Smallwood, UNITED KINGDOM
Green, Simon, Invergowrie, UNITED KINGDOM
Colyer, John, Bardsey, UNITED KINGDOM

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003079235	A1	20030424
APPLICATION INFO.:	US 2002-147354	A1	20020516 (10)
RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. WO 2000-GB4348, filed on 15 Nov 2000, UNKNOWN		

	NUMBER	DATE
PRIORITY INFORMATION:	GB 1999-27331	19991118
DOCUMENT TYPE:	Utility	
FILE SEGMENT:	APPLICATION	
LEGAL REPRESENTATIVE:	FROMMER LAWRENCE & HAUG, 745 FIFTH AVENUE- 10TH FL., NEW YORK, NY, 10151	
NUMBER OF CLAIMS:	22	
EXEMPLARY CLAIM:	1	

NUMBER OF DRAWINGS: 1 Drawing Page(s)

LINE COUNT: 2708

AB A method is provided for measuring in vivo in a transgenic non-human multicellular organism the activity of a cellular enzyme, which organism is transgenic by virtue of comprising one or more nucleic acid constructs encoding a binding domain and a binding partner thereof, wherein: (i) the binding domain and/or binding partner comprise a site subject to post-translational modification by the cellular enzyme; (ii) modification of the site by the enzyme affects the interaction between the binding domain and the binding partner; and (iii) the binding domain and the binding partner each comprise a detectable label such that when the binding domain and binding partner interact, a detectable physical characteristic of one or both of the labels is altered, which method comprises measuring the interaction between the binding domain and the binding partner by measuring changes in said physical characteristic in one or more cells of the transgenic organism. A transgenic non-human multicellular organism is also provided.

L3 ANSWER 2 OF 24 USPATFULL

ACCESSION NUMBER: 2003:100088 USPATFULL

TITLE: Treatment methods based on microcompetition for a limiting GABP complex

INVENTOR(S): Polansky, Hanan, Rochester, NY, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003069199	A1	20030410
APPLICATION INFO.:	US 2002-219334	A1	20020815 (10)
RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 2000-732360, filed on 7 Dec 2000, PENDING		
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	APPLICATION		
LEGAL REPRESENTATIVE:	Hanan Polansky, 3159 S. Winton Rd., Rochester, NY, 14623		
NUMBER OF CLAIMS:	26		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	28 Drawing Page(s)		
LINE COUNT:	14837		

AB Microcompetition for GABP between a foreign polynucleotide and a cellular GABP regulated gene is a risk factor associated with chronic disease such as obesity, cancer, atherosclerosis, stroke, osteoarthritis, diabetes, asthma, and other autoimmune diseases. The invention uses this novel discovery to present methods for the treatment of these chronic diseases. The methods are based on modifying such microcompetition, or the effect of such microcompetition on the cell. For instance, treatment may modify the cellular copy number of the foreign polynucleotide, change the rate of complex formation between GABP and either the foreign polynucleotide or the cellular GABP regulated gene, vary the expression of the cellular GABP regulated gene, or manipulate the activity of the gene product of the cellular GABP regulated gene. The invention also presents methods for treatment of chronic diseases resulting from other foreign polynucleotide-type disruptions.

L3 ANSWER 3 OF 24 USPATFULL

ACCESSION NUMBER: 2003:99511 USPATFULL

TITLE: Drug discovery assays based on microcompetition for a limiting GABP complex

INVENTOR(S): Polansky, Hanan, Rochester, NY, UNITED STATES

NUMBER	KIND	DATE
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PATENT INFORMATION: US 2003068616 A1 20030410
 APPLICATION INFO.: US 2002-223050 A1 20020814 (10)
 RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 2000-732360, filed
 on 7 Dec 2000, PENDING
 DOCUMENT TYPE: Utility
 FILE SEGMENT: APPLICATION
 LEGAL REPRESENTATIVE: Hanan Polansky, 3159 S. Winton Rd., Rochester, NY,
 14623
 NUMBER OF CLAIMS: 55
 EXEMPLARY CLAIM: 1
 NUMBER OF DRAWINGS: 28 Drawing Page(s)
 LINE COUNT: 14981

AB A recent discovery showed that microcompetition for GABP between a
 foreign polynucleotide and a cellular GABP regulated gene is a risk
 factor for some of the major chronic diseases, such as obesity, cancer,
 atherosclerosis, stroke, osteoarthritis, diabetes, asthma, and other
 autoimmune diseases. The invention uses this novel discovery to present
 assays for screening compounds based on their effectiveness in
 modulating such microcompetition, or the effects of such
 microcompetition on the cell. The selected compounds can be used in
 treatment of these chronic diseases. The invention also presents assays
 for screening compounds that can be used in treatment of chronic
 diseases resulting from other foreign polynucleotide-type disruptions.

L3 ANSWER 4 OF 24 USPATFULL

ACCESSION NUMBER: 2003:78501 USPATFULL
 TITLE: Nucleic acids, proteins, and antibodies
 INVENTOR(S): Rosen, Craig A., Laytonsville, MD, UNITED STATES
 Ruben, Steven M., Olney, MD, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003054421	A1	20030320
APPLICATION INFO.:	US 2002-102806	A1	20020322 (10)
RELATED APPLN. INFO.:	Continuation of Ser. No. US 2001-925298, filed on 10 Aug 2001, PENDING Continuation-in-part of Ser. No. WO 2000-US5881, filed on 8 Mar 2000, UNKNOWN		

	NUMBER	DATE
PRIORITY INFORMATION:	US 1999-124270P	19990312 (60)
DOCUMENT TYPE:	Utility	
FILE SEGMENT:	APPLICATION	
LEGAL REPRESENTATIVE:	HUMAN GENOME SCIENCES INC, 9410 KEY WEST AVENUE, ROCKVILLE, MD, 20850	
NUMBER OF CLAIMS:	24	
EXEMPLARY CLAIM:	1	
LINE COUNT:	20141	

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The present invention relates to novel ovarian cancer and/or breast
 cancer related polynucleotides, the polypeptides encoded by these
 polynucleotides herein collectively referred to as "ovarian and/or
 breast antigens," and antibodies that immunospecifically bind these
 polypeptides, and the use of such ovarian and/or breast polynucleotides,
 antigens, and antibodies for detecting, treating, preventing and/or
 prognosing disorders of the reproductive system, particularly disorders
 of the ovaries and/or breast, including, but not limited to, the
 presence of ovarian and/or breast cancer and ovarian and/or breast
 cancer metastases. More specifically, isolated ovarian and/or breast
 nucleic acid molecules are provided encoding novel ovarian and/or breast
 polypeptides. Novel ovarian and/or breast polypeptides and antibodies
 that bind to these polypeptides are provided. Also provided are vectors,
 host cells, and recombinant and synthetic methods for producing human

ovarian and/or breast polynucleotides, polypeptides, and/or antibodies. The invention further relates to diagnostic and therapeutic methods useful for diagnosing, treating, preventing and/or prognosing disorders related to the ovaries and/or breast, including ovarian and/or breast cancer, and therapeutic methods for treating such disorders. The invention further relates to screening methods for identifying agonists and antagonists of polynucleotides and polypeptides of the invention. The invention further relates to methods and/or compositions for inhibiting or promoting the production and/or function of the polypeptides of the invention.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L3 ANSWER 5 OF 24 USPATFULL

ACCESSION NUMBER: 2003:71403 USPATFULL
 TITLE: Protein fragment complementation assays for the detection of biological or drug interactions
 INVENTOR(S): Michnick, Stephen William Watson, Westmount, CANADA
 Pelletier, Joelle Nina, Westmount, CANADA
 Remy, Ingrid, Montreal, CANADA
 PATENT ASSIGNEE(S): Odyssey Pharmaceuticals, Inc., San Ramon, CA (non-U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003049688	A1	20030313
APPLICATION INFO.:	US 2002-154758	A1	20020524 (10)
RELATED APPLN. INFO.:	Continuation of Ser. No. US 2000-499464, filed on 7 Feb 2000, GRANTED, Pat. No. US 6428951 Continuation of Ser. No. US 1998-17412, filed on 2 Feb 1998, GRANTED, Pat. No. US 6270964		

	NUMBER	DATE
PRIORITY INFORMATION:	CA 1997-2196496	19970131
DOCUMENT TYPE:	Utility	
FILE SEGMENT:	APPLICATION	
LEGAL REPRESENTATIVE:	Isaac A. Angres, Suite 301, 2001 Jefferson Davis Highway, Arlington, VA, 22202	
NUMBER OF CLAIMS:	58	
EXEMPLARY CLAIM:	1	
NUMBER OF DRAWINGS:	7 Drawing Page(s)	
LINE COUNT:	2757	

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB We describe a strategy for designing and implementing protein-fragment complementation assays (PCAs) to detect biomolecular interactions in vivo and in vitro. The design, implementation and broad applications of this strategy are illustrated with a large number of enzymes with particular detail provided for the example of murine dihydrofolate reductase (DHFR). Fusion peptides consisting of N- and C-terminal fragments of murine DHFR fused to GCN4 leucine zipper sequences were coexpressed in Escherichia coli grown in minimal medium, where the endogenous DHFR activity was inhibited with trimethoprim. Coexpression of the complementary fusion products restored colony formation. Survival only occurred when both DHFR fragments were present and contained leucine-zipper forming sequences, demonstrating that reconstitution of enzyme activity requires assistance of leucine zipper formation. DHFR fragment-interface point mutants of increasing severity (Ile to Val, Ala and Gly) resulted in a sequential increase in E. coli doubling times illustrating the successful DHFR fragment reassembly rather than non-specific interactions between fragments. This assay could be used to study equilibrium and kinetic aspects of molecular interactions including protein-protein, protein-DNA, protein-RNA, protein-carbohydrate and protein-small molecule interactions, for

screening cDNA libraries for binding of a target protein with unknown proteins or libraries of small organic molecules for biological activity. The selection and design criteria applied here is developed for numerous examples of clonal selection, colorimetric, fluorometric and other assays based on enzymes whose products can be measured. The development of such assay systems is shown to be simple, and provides for a diverse set of protein fragment complementation applications.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L3 ANSWER 6 OF 24 USPATFULL

ACCESSION NUMBER: 2002:273550 USPATFULL
 TITLE: Nucleic acids, proteins and antibodies
 INVENTOR(S): Rosen, Craig A., Laytonsville, MD, UNITED STATES
 Ruben, Steven M., Olney, MD, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002151681	A1	20021017
APPLICATION INFO.:	US 2001-925300	A1	20010810 (9)
RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. WO 2000-US5988, filed on 8 Mar 2000, UNKNOWN		

	NUMBER	DATE
PRIORITY INFORMATION:	US 1999-124270P	19990312 (60)
DOCUMENT TYPE:	Utility	
FILE SEGMENT:	APPLICATION	
LEGAL REPRESENTATIVE:	HUMAN GENOME SCIENCES INC, 9410 KEY WEST AVENUE, ROCKVILLE, MD, 20850	
NUMBER OF CLAIMS:	23	
EXEMPLARY CLAIM:	1	
LINE COUNT:	29771	

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB This invention relates to newly identified prostate or prostate cancer related polynucleotides, the polypeptides encoded by these polynucleotides herein collectively referred to as "prostate cancer antigens," and to the complete gene sequences associated therewith and to the expression products thereof, and to antibodies that immunospecifically bind these polypeptides, as well as the use of such prostate cancer polynucleotides, antigens, and antibodies for detection, prevention, prognosis, and treatment of disorders of the reproductive system, particularly disorders of the prostate, including, but not limited to, the presence of prostate cancer and prostate cancer metastases. More specifically, isolated prostate cancer nucleic acid molecules are provided encoding novel prostate cancer polypeptides. Novel prostate cancer polypeptides and antibodies that bind to these polypeptides are provided. Also provided are vectors, host cells, and recombinant and synthetic methods for producing human prostate cancer polynucleotides, polypeptides, and/or antibodies. The invention further relates to diagnostic and therapeutic methods useful for diagnosing, treating, preventing and/or prognosing disorders related to the prostate, including prostate cancer, and therapeutic methods for treating such disorders. The invention further relates to screening methods for identifying agonists and antagonists of polynucleotides and polypeptides of the invention. The invention further relates to methods and/or compositions for inhibiting or promoting the production and/or function of the polypeptides of the invention.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L3 ANSWER 7 OF 24 USPATFULL

ACCESSION NUMBER: 2002:272900 USPATFULL
 TITLE: Stimulus-inducible protein kinase complex and methods

INVENTOR(S): of use therefor
Mercurio, Frank, San Diego, CA, UNITED STATES
Zhu, Hengyi, San Diego, CA, UNITED STATES
Barbosa, Miguel, San Diego, CA, UNITED STATES
Li, Jian Wu, San Diego, CA, UNITED STATES
Murray, Brion W., San Diego, CA, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002151021	A1	20021017
APPLICATION INFO.:	US 2001-844908	A1	20010427 (9)
RELATED APPLN. INFO.:	Division of Ser. No. US 1997-910820, filed on 13 Aug 1997, PATENTED Continuation-in-part of Ser. No. US 1996-697393, filed on 26 Aug 1996, PATENTED		
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	APPLICATION		
LEGAL REPRESENTATIVE:	Timothy X. Gibson, Mathews, Collins, Shepherd & Gould, Suite 306, 100 Thanet Circle, Princeton, NJ, 08540		
NUMBER OF CLAIMS:	32		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	31 Drawing Page(s)		
LINE COUNT:	2343		

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Compositions and methods are provided for treating NF-.kappa.B-related conditions. In particular, the invention provides a stimulus-inducible IKK signalsome, and components and variants thereof. An IKK signalsome or component thereof may be used, for example, to identify antibodies and other modulating agents that inhibit or activate signal transduction via the NF-.kappa.B cascade. IKK signalsome, components thereof and/or modulating agents may also be used for the treatment of diseases associated with NF-.kappa.B activation.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L3 ANSWER 8 OF 24 USPATFULL
ACCESSION NUMBER: 2002:258894 USPATFULL
TITLE: 38646, a novel guanine nucleotide exchange factor and uses therefor
INVENTOR(S): Glucksmann, Maria Alexandra, Lexington, MA, UNITED STATES
PATENT ASSIGNEE(S): Millennium Pharmaceuticals, Inc., Cambridge, MA (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002142464	A1	20021003
APPLICATION INFO.:	US 2001-950491	A1	20010910 (9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-231089P	20000908 (60)
DOCUMENT TYPE:	Utility	
FILE SEGMENT:	APPLICATION	
LEGAL REPRESENTATIVE:	AKIN, GUMP, STRAUSS, HAUER & FELD, L.L.P., ONE COMMERCE SQUARE, 2005 MARKET STREET, SUITE 2200, PHILADELPHIA, PA, 19103	
NUMBER OF CLAIMS:	22	
EXEMPLARY CLAIM:	1	
NUMBER OF DRAWINGS:	15 Drawing Page(s)	
LINE COUNT:	4625	

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The invention provides isolated nucleic acids molecules, designated 38646 nucleic acid molecules, which encode a novel guanine-nucleotide exchange factor. The invention also provides antisense nucleic acid

molecules, recombinant expression vectors containing 38646 nucleic acid molecules, host cells into which the expression vectors have been introduced, and non-human transgenic animals in which a 38646 gene has been introduced or disrupted. The invention still further provides isolated 38646 proteins, fusion proteins, antigenic peptides and anti-38646 antibodies. Diagnostic methods utilizing compositions of the invention are also provided. 38646 expression and activity can be modulated to affect cell shape, motility, cytoskeleton organization, and intracellular protein and vesicle localization or to affect the tensile strength or integrity of a tissue.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L3 ANSWER 9 OF 24 USPATFULL

ACCESSION NUMBER: 2002:251221 USPATFULL
 TITLE: ASIP-related proteins
 INVENTOR(S): Reddy, Roopa, Sunnyvale, CA, UNITED STATES
 Tang, Y. Tom, San Jose, CA, UNITED STATES
 Baughn, Mariah R., San Leandro, CA, UNITED STATES
 Krasnow, Randi E., Stanford, CA, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002137166	A1	20020926
APPLICATION INFO.:	US 2001-757781	A1	20010109 (9)
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	APPLICATION		
LEGAL REPRESENTATIVE:	INCYTE GENOMICS, INC., 3160 PORTER DRIVE, PALO ALTO, CA, 94304		
NUMBER OF CLAIMS:	22		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	40 Drawing Page(s)		
LINE COUNT:	3608		

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The invention provides cDNAs which encode ASIP-related proteins. It also provides for the use of the cDNAs, fragments, complements, and variants thereof and of the encoded proteins, portions thereof and antibodies thereto for diagnosis and treatment of cancer, particularly bladder transitional cell carcinoma. The invention additionally provides expression vectors and host cells for the production of the protein and a transgenic model system.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L3 ANSWER 10 OF 24 USPATFULL

ACCESSION NUMBER: 2002:227650 USPATFULL
 TITLE: Integrin-linked kinase and its uses
 INVENTOR(S): Dedhar, Shoukat, Vancouver, CANADA
 Hannigan, Greg, Toronto, CANADA

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002122801	A1	20020905
APPLICATION INFO.:	US 2001-840704	A1	20010423 (9)
RELATED APPLN. INFO.:	Continuation of Ser. No. US 2000-566906, filed on 9 May 2000, GRANTED, Pat. No. US 6369205 Division of Ser. No. US 1999-390425, filed on 3 Sep 1999, GRANTED, Pat. No. US 6338958 Continuation-in-part of Ser. No. US 1997-955841, filed on 21 Oct 1997, GRANTED, Pat. No. US 6013782 Continuation-in-part of Ser. No. US 1996-752345, filed on 19 Nov 1996, ABANDONED		

NUMBER	DATE

PRIORITY INFORMATION: US 1995-9074P 19951221 (60)
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: Pamela J. Sherwood, Bozicevic, Field & Francis LLP,
Suite 200, 200 Middlefield Road, Menlo Park, CA, 94024
NUMBER OF CLAIMS: 18
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 23 Drawing Page(s)
LINE COUNT: 3236

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Methods for isolating ILK genes are provided. The ILK nucleic acid compositions find use in identifying homologous or related proteins and the DNA sequences encoding such proteins; in producing compositions that modulate the expression or function of the protein; and in studying associated physiological pathways. In addition, modulation of the gene activity in vivo is used for prophylactic and therapeutic purposes, such as identification of cell type based on expression, and the like.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L3 ANSWER 11 OF 24 USPATFULL

ACCESSION NUMBER: 2002:72627 USPATFULL
TITLE: Nucleic, acids, proteins, and antibodies
INVENTOR(S): Rosen, Craig A., Laytonsville, MD, UNITED STATES
Ruben, Steven M., Olney, MD, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002039764	A1	20020404
APPLICATION INFO.:	US 2001-925298	A1	20010810 (9)
RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. WO 2000-US5881, filed on 8 Mar 2000, UNKNOWN		

	NUMBER	DATE
PRIORITY INFORMATION:	US 1999-124270P	19990312 (60)
DOCUMENT TYPE:	Utility	
FILE SEGMENT:	APPLICATION	
LEGAL REPRESENTATIVE:	HUMAN GENOME SCIENCES INC, 9410 KEY WEST AVENUE, ROCKVILLE, MD, 20850	
NUMBER OF CLAIMS:	23	
EXEMPLARY CLAIM:	1	
LINE COUNT:	20087	

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The present invention relates to novel ovarian cancer and/or breast cancer related polynucleotides, the polypeptides encoded by these polynucleotides herein collectively referred to as "ovarian and/or breast antigens," and antibodies that immunospecifically bind these polypeptides, and the use of such ovarian and/or breast polynucleotides, antigens, and antibodies for detecting, treating, preventing and/or prognosing disorders of the reproductive system, particularly disorders of the ovaries and/or breast, including, but not limited to, the presence of ovarian and/or breast cancer and ovarian and/or breast cancer metastases. More specifically, isolated ovarian and/or breast nucleic acid molecules are provided encoding novel ovarian and/or breast polypeptides. Novel ovarian and/or breast polypeptides and antibodies that bind to these polypeptides are provided. Also provided are vectors, host cells, and recombinant and synthetic methods for producing human ovarian and/or breast polynucleotides, polypeptides, and/or antibodies. The invention further relates to diagnostic and therapeutic methods useful for diagnosing, treating, preventing and/or prognosing disorders related to the ovaries and/or breast, including ovarian and/or breast cancer, and therapeutic methods for treating such disorders. The invention further relates to screening methods for identifying agonists

and antagonists of polynucleotides and polypeptides of the invention. The invention further relates to methods and/or compositions for inhibiting or promoting the production and/or function of the polypeptides of the invention.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L3 ANSWER 12 OF 24 USPATFULL

ACCESSION NUMBER: 2002:32181 USPATFULL

TITLE: Methods of monitoring enzyme activity

INVENTOR(S): Griffiths, Gary, Oldham, UNITED KINGDOM

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002019002	A1	20020214
APPLICATION INFO.:	US 2001-877919	A1	20010607 (9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-211313P	20000613 (60)
DOCUMENT TYPE:	Utility	
FILE SEGMENT:	APPLICATION	
LEGAL REPRESENTATIVE:	PALMER & DODGE, LLP, ONE BEACON STREET, BOSTON, MA, 02108-3190	
NUMBER OF CLAIMS:	40	
EXEMPLARY CLAIM:	1	
LINE COUNT:	3633	

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB We describe a method for monitoring the activity of an enzyme, the method comprising the steps of: providing a binding domain which includes a site for enzymatic modification; providing a binding partner which binds to the binding domain in a manner which is dependent upon modification of the site. The binding domain is contacted with the enzyme; and binding of the binding domain to the binding partner is detected as an indication of the activity of the enzyme. One of the binding domain and binding partner comprises a polypeptide and the other of the binding domain and binding partner comprises a nucleic acid.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L3 ANSWER 13 OF 24 USPATFULL

ACCESSION NUMBER: 2002:12284 USPATFULL

TITLE: Arrayed transfection method and uses related thereto

INVENTOR(S): Sabatini, David M., Cambridge, MA, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002006664	A1	20020117
APPLICATION INFO.:	US 2001-817003	A1	20010322 (9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-193580P	20000330 (60)
	US 1999-154737P	19990917 (60)
DOCUMENT TYPE:	Utility	
FILE SEGMENT:	APPLICATION	
LEGAL REPRESENTATIVE:	ROPES & GRAY, ONE INTERNATIONAL PLACE, BOSTON, MA, 02110-2624	
NUMBER OF CLAIMS:	37	
EXEMPLARY CLAIM:	1	
NUMBER OF DRAWINGS:	7 Drawing Page(s)	
LINE COUNT:	2671	

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB An arrayed transfection method of introducing nucleic acid of interest

into cells.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L3 ANSWER 14 OF 24 USPATFULL

ACCESSION NUMBER: 2002:75564 USPATFULL
TITLE: Integrin-linked kinase and its uses
INVENTOR(S): Dedhar, Shoukat, Vancouver, CANADA
Hannigan, Greg, Ontario, CANADA
PATENT ASSIGNEE(S): Sunnybrook Health Science Centre, Toronto, CANADA
(non-U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6369205	B1	20020409
APPLICATION INFO.:	US 2000-566906		20000509 (9)
RELATED APPLN. INFO.:	Division of Ser. No. US 1999-390425, filed on 3 Sep 1999 Continuation-in-part of Ser. No. US 1997-955841, filed on 21 Oct 1997, now patented, Pat. No. US 6013782 Continuation-in-part of Ser. No. US 1996-752345, filed on 19 Nov 1996, now abandoned		

	NUMBER	DATE
PRIORITY INFORMATION:	US 1995-9074P	19951221 (60)
DOCUMENT TYPE:	Utility	
FILE SEGMENT:	GRANTED	
PRIMARY EXAMINER:	Clark, Deborah J. R.	
ASSISTANT EXAMINER:	Chen, Shin-Lin	
LEGAL REPRESENTATIVE:	Sherwood, Pamela J., Bozicevic, Field & Francis LLP	
NUMBER OF CLAIMS:	5	
EXEMPLARY CLAIM:	1	
NUMBER OF DRAWINGS:	25 Drawing Figure(s); 23 Drawing Page(s)	
LINE COUNT:	3200	

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Methods for isolating ILK genes are provided, The ILK nucleic acid compositions find use in identifying homologous or related proteins and the DNA sequences encoding such proteins; in producing compositions that modulate the expression or function of the protein; and in studying associated physiological pathways. In addition, modulation of the gene activity in vivo is used for prophylactic and therapeutic purposes, such as identification of cell type based on expression, and the like.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L3 ANSWER 15 OF 24 USPATFULL

ACCESSION NUMBER: 2002:9751 USPATFULL
TITLE: Integrin-linked kinase and its uses
INVENTOR(S): Dedhar, Shoukat, Vancouver, CANADA
Hannigan, Greg, Ontario, CANADA
PATENT ASSIGNEE(S): Sunnybrook Health Science Centre, Toronto, CANADA
(non-U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6338958	B1	20020115
APPLICATION INFO.:	US 1999-390425		19990903 (9)
RELATED APPLN. INFO.:	Continuation of Ser. No. US 1998-35706, filed on 5 Mar 1998, now patented, Pat. No. US 6001622 Continuation-in-part of Ser. No. US 1997-955841, filed on 21 Oct 1997, now patented, Pat. No. US 6013782 Continuation-in-part of Ser. No. US 1996-752345, filed on 19 Nov 1996, now abandoned		

	NUMBER	DATE
PRIORITY INFORMATION:	US 1995-9074P	19951221 (60)
DOCUMENT TYPE:	Utility	
FILE SEGMENT:	GRANTED	
PRIMARY EXAMINER:	Clark, Deborah J. R.	
ASSISTANT EXAMINER:	Chen, Shin-Lin	
LEGAL REPRESENTATIVE:	Sherwood, Pamela J., Bozicevic, Field & Francis LLP	
NUMBER OF CLAIMS:	4	
EXEMPLARY CLAIM:	1	
NUMBER OF DRAWINGS:	21 Drawing Figure(s); 23 Drawing Page(s)	
LINE COUNT:	3203	

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Methods for isolating ILK genes are provided. The ILK nucleic acid compositions find use in identifying homologous or related proteins and the DNA sequences encoding such proteins; in producing compositions that modulate the expression or function of the protein; and in studying associated physiological pathways. In addition, modulation of the gene activity in vivo is used for prophylactic and therapeutic purposes, such as identification of cell type based on expression, and the like.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L3 ANSWER 16 OF 24 USPATFULL

ACCESSION NUMBER: 2001:116764 USPATFULL
 TITLE: Ataxia-telangiectasia gene and its genomic organization
 INVENTOR(S): Shiloh, Yosef, Tel Aviv, Israel
 PATENT ASSIGNEE(S): Ramot-University Authority for Applied Research and Industrial Development, Tel Aviv, Israel (non-U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6265158	B1	20010724
	WO 9636691		19961121
APPLICATION INFO.:	US 1998-952014		19980202 (8)
	WO 1996-US7025		19960516
			19980202 PCT 371 date
			19980202 PCT 102(e) date
RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 1996-629001, filed on 8 Apr 1996, now patented, Pat. No. US 5858661		
	Continuation-in-part of Ser. No. US 1995-441822, filed on 16 May 1995, now patented, Pat. No. US 5756288		
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	GRANTED		
PRIMARY EXAMINER:	Jones, W. Gary		
ASSISTANT EXAMINER:	Goldberg, Jeanine		
LEGAL REPRESENTATIVE:	Kohn & Associates		
NUMBER OF CLAIMS:	7		
EXEMPLARY CLAIM:	1,7		
NUMBER OF DRAWINGS:	4 Drawing Figure(s); 2 Drawing Page(s)		
LINE COUNT:	3109		

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB A purified and isolated gene, designated ATM, mutations of which cause ataxia-telangiectasia, its genomic organization, methods for the detection of the defective gene, the purified polypeptide encoded by the defective gene, and antibodies recognizing the defective protein.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L3 ANSWER 17 OF 24 USPATFULL

ACCESSION NUMBER: 2001:107664 USPATFULL
 TITLE: Stimulus-inducible protein kinase complex and methods of use therefor

INVENTOR(S): Mercurio, Frank, San Diego, CA, United States
 Zhu, Hengyi, San Diego, CA, United States
 Barbosa, Miguel, San Diego, CA, United States
 Li, Jian Wu, San Diego, CA, United States
 Murray, Brion W., San Diego, CA, United States
 PATENT ASSIGNEE(S): Signal Pharmaceuticals, Inc., San Diego, CA, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6258579	B1	20010710
APPLICATION INFO.:	US 1997-910820		19970813 (8)
RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 1996-697393, filed on 26 Aug 1996, now patented, Pat. No. US 5972674		
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	GRANTED		
PRIMARY EXAMINER:	Patterson, Jr., Charles L.		
LEGAL REPRESENTATIVE:	Mathews, Collins, Shepherd & Gould, P.A.		
NUMBER OF CLAIMS:	4		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	30 Drawing Figure(s); 28 Drawing Page(s)		
LINE COUNT:	1713		

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Compositions and methods are provided for treating NF- κ B-related conditions. In particular, the invention provides a stimulus-inducible IKK signalsome, and components and variants thereof. An IKK signalsome or component thereof may be used, for example, to identify antibodies and other modulating agents that inhibit or activate signal transduction via the NF- κ B cascade. IKK signalsome, components thereof and/or modulating agents may also be used for the treatment of diseases associated with NF- κ B activation.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L3 ANSWER 18 OF 24 USPATFULL

ACCESSION NUMBER: 2001:48208 USPATFULL
 TITLE: Ataxia-telangiectasia gene
 INVENTOR(S): Shiloh, Yosef, Tel Aviv, Israel
 Tagle, Danilo A., Gaithersburg, MD, United States
 Collins, Francis, Rockville, MD, United States
 PATENT ASSIGNEE(S): The United States of America as represented by the Department of Health and Human Services, Washington, DC, United States (U.S. government)
 Ramot University Authority for Applied Research and Industrial Dev., Israel (non-U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6211336	B1	20010403
	WO 9636695		19961121
APPLICATION INFO.:	US 1998-952127		19980226 (8)
	WO 1996-US7040		19960516
			19980226 PCT 371 date
			19980226 PCT 102(e) date
RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 1995-508836, filed on 28 Jul 1995, now patented, Pat. No. US 5777093		
	Continuation-in-part of Ser. No. US 1995-493092, filed on 21 Jun 1995, now patented, Pat. No. US 5728807		
	Continuation-in-part of Ser. No. US 1995-441822, filed on 16 May 1995, now patented, Pat. No. US 5756288		
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	Granted		
PRIMARY EXAMINER:	Cintins, Marianne M.		
ASSISTANT EXAMINER:	Delacroix-Muirheid, C.		

LEGAL REPRESENTATIVE: Kohn & Associates
NUMBER OF CLAIMS: 5
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 10 Drawing Figure(s); 4 Drawing Page(s)
LINE COUNT: 2279

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB There is provided a purified amino acid sequence selected from the group of Sequence ID No.: 3 and analogs thereof and mutations of Sequence ID No.: 3 which cause ataxia-telangiectasia. Also provided is a purified amino acid sequence as set forth in Sequence ID No.: 3 and analogs thereof.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L3 ANSWER 19 OF 24 USPATFULL

ACCESSION NUMBER: 2001:36598 USPATFULL
TITLE: Mutated forms of the ataxia-telangiectasia gene and method to screen for a partial A-T phenotype
INVENTOR(S): Shiloh, Yosef, Tel Aviv, Israel
PATENT ASSIGNEE(S): Ramot-University Authority for Applied Research and Industrial Development Ltd., Tel Aviv, Israel (non-U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6200749	B1	20010313
APPLICATION INFO.:	US 1996-642274		19960503 (8)
RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 1996-629001, filed on 8 Apr 1996 Continuation-in-part of Ser. No. US 1995-441822, filed on 16 May 1995, now patented, Pat. No. US 5756288		

DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Arthur, Lisa B.
LEGAL REPRESENTATIVE: Kohn & Associates
NUMBER OF CLAIMS: 5
EXEMPLARY CLAIM: 1,4
NUMBER OF DRAWINGS: 8 Drawing Figure(s); 2 Drawing Page(s)
LINE COUNT: 3090

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB A purified and isolated gene, designated ATM, is described mutations of which cause ataxia-telangiectasia and its genomic organization.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L3 ANSWER 20 OF 24 USPATFULL

ACCESSION NUMBER: 2000:164282 USPATFULL
TITLE: Serine/threonine protein kinases
INVENTOR(S): Bandman, Olga, Mountain View, CA, United States
Tang, Y. Tom, San Jose, CA, United States
Goli, Surya K., San Jose, CA, United States
Corley, Neil C., Mountain View, CA, United States
Guegler, Karl J., Menlo Park, CA, United States
Gorgone, Gina A., Boulder Creek, CA, United States
Hillman, Jennifer L., Mountain View, CA, United States
PATENT ASSIGNEE(S): Incyte Pharmaceuticals, Inc., Palo Alto, CA, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6156523		20001205
APPLICATION INFO.:	US 1998-153939		19980916 (9)
RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 1996-749902, filed on 15 Nov 1996, now patented, Pat. No. US 5985635		

DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Mertz, Prema
ASSISTANT EXAMINER: Murphy, Joseph F.
LEGAL REPRESENTATIVE: Incyte Pharmaceuticals, Inc., Murry, Lynn E.
NUMBER OF CLAIMS: 5
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 14 Drawing Figure(s); 14 Drawing Page(s)
LINE COUNT: 2733

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The invention provides human serine/threonine protein kinases (HSTK) and polynucleotides which identify and encode HSTK. The invention also provides expression vectors, host cells, antibodies, agonists, and antagonists. The invention also provides methods for diagnosing, treating or preventing disorders associated with expression of HSTK.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L3 ANSWER 21 OF 24 USPATFULL

ACCESSION NUMBER: 1999:163477 USPATFULL
TITLE: Integrin-linked kinase and its use
INVENTOR(S): Dedhar, Shoukat, Vancouver, Canada
Hannigan, Greg, Ontario, Canada
PATENT ASSIGNEE(S): Sunnybrook Health Science Centre, Ontario, Canada
(non-U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6001622		19991214
APPLICATION INFO.:	US 1998-35706		19980305 (9)
RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 1997-955841, filed on 21 Oct 1997 which is a continuation-in-part of Ser. No. US 1996-752345, filed on 19 Nov 1996		

	NUMBER	DATE
PRIORITY INFORMATION:	US 1995-9074P	19951221 (60)
DOCUMENT TYPE:	Utility	
FILE SEGMENT:	Granted	
PRIMARY EXAMINER:	Achutamurthy, Ponnathapu	
LEGAL REPRESENTATIVE:	Bozicevic, Field & Francis LLP, Sherwood, Pamela	
NUMBER OF CLAIMS:	4	
EXEMPLARY CLAIM:	1	
NUMBER OF DRAWINGS:	21 Drawing Figure(s); 23 Drawing Page(s)	
LINE COUNT:	3148	

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Methods for isolating ILK genes are provided. The ILK nucleic acid compositions find use in identifying homologous or related proteins and the DNA sequences encoding such proteins; in producing compositions that modulate the expression or function of the protein; and in studying associated physiological pathways. In addition, modulation of the gene activity in vivo is used for prophylactic and therapeutic purposes, such as identification of cell type based on expression, and the like.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L3 ANSWER 22 OF 24 USPATFULL

ACCESSION NUMBER: 1999:146335 USPATFULL
TITLE: Nucleic acids encoding novel human serine/threonine protein kinases
INVENTOR(S): Bandman, Olga, Mountain View, CA, United States
Goli, Surya K., Sunnyvale, CA, United States
Hillman, Jennifer L., San Jose, CA, United States
PATENT ASSIGNEE(S): Incyte Pharmaceuticals, Inc., Palo Alto, CA, United

States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5985635		19991116
APPLICATION INFO.:	US 1996-749902		19961115 (8)
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	Granted		
PRIMARY EXAMINER:	Teng, Sally P.		
LEGAL REPRESENTATIVE:	Incyte Pharmaceuticals, Inc.		
NUMBER OF CLAIMS:	6		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	12 Drawing Figure(s); 12 Drawing Page(s)		
LINE COUNT:	2520		

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The present invention provides human serine/threonine kinase (HSTK) and polynucleotides which identify and encode HSTK. The invention also provides genetically engineered expression vectors and host cells comprising the nucleic acid sequences encoding HSTK and a method for producing HSTK. The invention also provides for use of HSTK and agonists, antibodies, or antagonists specifically binding HSTK, in the prevention and treatment of diseases associated with expression of HSTK. Additionally, the invention provides for the use of antisense molecules to polynucleotides encoding HSTK for the treatment of diseases associated with the expression of HSTK. The invention also provides diagnostic assays which utilize the polynucleotide, or fragments or the complement thereof, and antibodies specifically binding HSTK.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L3 ANSWER 23 OF 24 USPATFULL

ACCESSION NUMBER: 1999:132563 USPATFULL
TITLE: Stimulus-inducible protein kinase complex and methods of use therefor
INVENTOR(S): Mercurio, Frank, San Diego, CA, United States
Zhu, Hengyi, San Diego, CA, United States
Barbosa, Miguel, San Diego, CA, United States
PATENT ASSIGNEE(S): Signal Pharmaceuticals, Inc., San Diego, CA, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5972674		19991026
APPLICATION INFO.:	US 1996-697393		19960826 (8)
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	Granted		
PRIMARY EXAMINER:	Chan, Christina Y.		
ASSISTANT EXAMINER:	Nolan, Patrick J.		
LEGAL REPRESENTATIVE:	SEED and BERRY LLP		
NUMBER OF CLAIMS:	2		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	11 Drawing Figure(s); 6 Drawing Page(s)		
LINE COUNT:	945		

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Compositions and methods are provided for treating NF-.kappa.B-related conditions. In particular, the invention provides a stimulus-inducible I.kappa.B.alpha. kinase complex, and components and variants thereof. I.kappa.B.alpha. kinase complex may be used, for example, to identify antibodies and other agents that inhibit or activate signal transduction via the NF-.kappa.B cascade. I.kappa.B.alpha. kinase complex, components thereof and/or such agents may also be used for the treatment of diseases associated with NF-.kappa.B activation.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L3 ANSWER 24 OF 24 USPATFULL

ACCESSION NUMBER: 1999:4329 USPATFULL
TITLE: Ataxia-telangiectasia gene and its genomic organization
INVENTOR(S): Shiloh, Yosef, Tel Aviv, Israel
PATENT ASSIGNEE(S): RAMOT-University Authority for Applied Research and
Industrial Development, Tel Aviv, Israel (non-U.S.
corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5858661		19990112
APPLICATION INFO.:	US 1996-629001		19960408 (8)
RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 1995-441822, filed on 16 May 1995, now patented, Pat. No. US 5756288		
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	Granted		
PRIMARY EXAMINER:	Arthur, Lisa B.		
LEGAL REPRESENTATIVE:	Kohn & Associates		
NUMBER OF CLAIMS:	9		
EXEMPLARY CLAIM:	1,7		
NUMBER OF DRAWINGS:	6 Drawing Figure(s); 2 Drawing Page(s)		
LINE COUNT:	3461		

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB A purified and isolated gene, designated ATM, mutations of which cause ataxia-telangiectasia and its genomic organization is disclosed. Methods and a kit for the detection of carriers of mutations of the ATM gene are provided by analysis of nucleic acids isolated from patients including in situ hybridization, Northern blotting and reverse transcriptase--polymerase chain reaction, Southern blotting, single strand conformational polymorphism, restriction endonuclease fingerprinting (REF), PCR amplification and DNA-chip analysis.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

=> d l3 ibib kxic tot
'KXIC' IS NOT A VALID FORMAT FOR FILE 'USPATFULL'

The following are valid formats:

The default display format is STD.

ABS ----- AB
ALL ----- AN, TI, IN, INA, PA, PAA, PAT, PI, AI, PTERM, DCD,
RLI, PRAI, DT, FS, REP, REN, EXNAM, LREP, CLMN, ECL,
DRWN, AB, GOVI, PARN, SUMM, DRWD, DETD, CLM, INCL,
INCLM, INCLS, NCL, NCLM, NCLS, IC, ICM, ICS,
EXF, ARTU
ALLG ----- ALL plus PAGE.DRAW
BIB ----- AN, TI, IN, INA, PA, PAA, PAT, PI, AI, PTERM, DCD, RLI,
PRAI, DT, FS, EXNAM, LREP, CLMN, ECL, DRWN, LN.CNT
BIB.EX ----- BIB for original and latest publication
BIBG ----- BIB plus PAGE.DRAW
BROWSE ----- See "HELP BROWSE" or "HELP DISPLAY BROWSE". BROWSE must
entered on the same line as DISPLAY, e.g., D BROWSE.
CAS ----- OS, CC, SX, ST, IT
CBIB ----- AN, TI, IN, INA, PA, PAA, PAT, PI, AI, PRAI, DT, FS
DALL ----- ALL, delimited for post-processing
FP ----- PI, TI, IN, INA, PA, PAA, PAT, PTERM, DCD, AI, RLI,
PRAI, IC, ICM, ICS, INCL, INCLM, INCLS, NCL,
NCLM, NCLS, EXF, REP, REN, ARTU, EXNAM, LREP,
CLMN, DRWN, AB
FP.EX ----- FP for original and latest publication

FPALL ----- PI, TI, IN, INA, PA, PAA, PAT, PETRM, DCD, AI,
 RLI, PRAI, IC, ICM, ICS, INCL, INCLM, INCLS, NCL, NCLM,
 NCLS, EXF, REP, REN, ARTU, EXNAM, LREP, CLMN, DRWN, AB,
 PARN, SUMM, DRWD, DETD, CLM
 FPBIB ----- PI, TI, IN, INA, PA, PAA, PAT, PTERM, DCD, AI,
 RLI, PRAI, REP, REN, EXNAM, LREP, CLM, CLMN, DRWN
 FHITSTR ---- HIT RN, its text modification, its CA index name, and
 its structure diagram
 FPG ----- FP plus PAGE.DRAW
 GI ----- PN and page image numbers
 HIT ----- All fields containing hit terms
 HITRN ----- HIT RN and its text modification
 HITSTR ----- HIT RN, its text modification, its CA index name, and
 its structure diagram
 IABS ----- ABS, indented with text labels
 IALL ----- ALL, indented with text labels
 IALLG ----- IALL plus PAGE.DRAW
 IBIB ----- BIB, indented with text labels
 IBIB.EX ---- IBIB for original and latest publication
 IBIBG ----- IBIB plus PAGE.DRAW
 IMAX ----- MAX, indented with text labels
 IMAX.EX ---- IMAX for original and latest publication
 IND ----- INCL, INCLM, INCLS, NCL, NCLM, NCLS, IC, ICM, ICS,
 EXF, ARTU, OS, CC, SX, ST, IT
 ISTD ----- STD, indented with text labels
 KWIC ----- All hit terms plus 20 words on either side
 MAX ----- AN, TI, IN, INA, PA, PAA, PAT, PI, AI, PTERM, DCD,
 RLI, PRAI, DT, FS, REP, REN, EXNAM, LREP, CLMN, ECL,
 DRWN, AB, GOVI, PARN, SUMM, DRWD, DETD, CLM, INCL,
 INCLM, INCLS, NCL, NCLM, NCLS, IC, ICM, ICS,
 EXF, ARTU OS, CC, SX, ST, IT
 MAX.EX ----- MAX for original and latest publication
 OCC ----- List of display fields containing hit terms
 SBIB ----- AN, TI, IN, INA, PA, PAA, PAT, PI, AI, RLI, PRAI,
 DT, FS, LN.CNT
 SCAN ----- AN, TI, NCL, NCLM, NCLS, IC, ICM, ICS (random display
 without answer number. SCAN must be entered on the
 same line as DISPLAY, e.g., D SCAN)
 STD ----- AN, TI, IN, INA, PA, PAA, PAT, PI, AI, RLI, PRAI,
 DT, FS, LN.CNT, INCL, INCLM, INCLS, NCL, NCLM, NCLS,
 IC, ICM, ICS, EXF (STD is the default)
 STD.EX ----- STD for original and latest publication
 TRIAL ----- AN, TI, INCL, INCLM, INCLS, NCL, NCLM, NCLS, IC,
 ICM, ICS

ENTER DISPLAY FORMAT (STD):
 ENTER DISPLAY FORMAT (STD):std

L3 ANSWER 1 OF 24 USPATFULL
 AN 2003:114491 USPATFULL
 TI Assay for measuring enzyme activity in vivo
 IN Craig, Roger Kingdon, Smallwood, UNITED KINGDOM
 Green, Simon, Invergowrie, UNITED KINGDOM
 Colyer, John, Bardsey, UNITED KINGDOM
 PI US 2003079235 A1 20030424
 AI US 2002-147354 A1 20020516 (10)
 RLI Continuation-in-part of Ser. No. WO 2000-GB4348, filed on 15 Nov 2000,
 UNKNOWN
 PRAI GB 1999-27331 19991118
 DT Utility
 FS APPLICATION
 LN.CNT 2708
 INCL INCLM: 800/003.000
 INCLS: 435/006.000; 424/009.600

NCL NCLM: 800/003.000
 NCLS: 435/006.000; 424/009.600
 IC [7]
 ICM: A61K049-00
 ICS: G01N033-00; C12Q001-68

L3 ANSWER 2 OF 24 USPATFULL
 AN 2003:100088 USPATFULL
 TI Treatment methods based on microcompetition for a limiting GABP complex
 IN Polansky, Hanan, Rochester, NY, UNITED STATES
 PI US 2003069199 A1 20030410
 AI US 2002-219334 A1 20020815 (10)
 RLI Continuation-in-part of Ser. No. US 2000-732360, filed on 7 Dec 2000,
 PENDING
 DT Utility
 FS APPLICATION
 LN.CNT 14837
 INCL INCLM: 514/044.000
 INCLS: 424/093.200; 424/186.100
 NCL NCLM: 514/044.000
 NCLS: 424/093.200; 424/186.100
 IC [7]
 ICM: A61K048-00
 ICS: A61K039-12

L3 ANSWER 3 OF 24 USPATFULL
 AN 2003:99511 USPATFULL
 TI Drug discovery assays based on microcompetition for a limiting GABP
 complex
 IN Polansky, Hanan, Rochester, NY, UNITED STATES
 PI US 2003068616 A1 20030410
 AI US 2002-223050 A1 20020814 (10)
 RLI Continuation-in-part of Ser. No. US 2000-732360, filed on 7 Dec 2000,
 PENDING
 DT Utility
 FS APPLICATION
 LN.CNT 14981
 INCL INCLM: 435/005.000
 INCLS: 435/007.210; 435/456.000; 435/320.100; 435/325.000; 435/366.000
 NCL NCLM: 435/005.000
 NCLS: 435/007.210; 435/456.000; 435/320.100; 435/325.000; 435/366.000
 IC [7]
 ICM: C12Q001-70
 ICS: G01N033-567; C12N015-86; C12N005-08

L3 ANSWER 4 OF 24 USPATFULL
 AN 2003:78501 USPATFULL
 TI Nucleic acids, proteins, and antibodies
 IN Rosen, Craig A., Laytonsville, MD, UNITED STATES
 Ruben, Steven M., Olney, MD, UNITED STATES
 PI US 2003054421 A1 20030320
 AI US 2002-102806 A1 20020322 (10)
 RLI Continuation of Ser. No. US 2001-925298, filed on 10 Aug 2001, PENDING
 Continuation-in-part of Ser. No. WO 2000-US5881, filed on 8 Mar 2000,
 UNKNOWN
 PRAI US 1999-124270P 19990312 (60)
 DT Utility
 FS APPLICATION
 LN.CNT 20141
 INCL INCLM: 435/007.230
 INCLS: 435/006.000; 435/069.100; 435/320.100; 435/325.000; 435/183.000;
 536/023.200
 NCL NCLM: 435/007.230
 NCLS: 435/006.000; 435/069.100; 435/320.100; 435/325.000; 435/183.000;

536/023.200

IC [7]
ICM: C12Q001-68
ICS: G01N033-574; C07H021-04; C12N009-00; C12P021-02; C12N005-06
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L3 ANSWER 5 OF 24 USPATFULL
AN 2003:71403 USPATFULL
TI Protein fragment complementation assays for the detection of biological
or drug interactions
IN Michnick, Stephen William Watson, Westmount, CANADA
Pelletier, Joelle Nina, Westmount, CANADA
Remy, Ingrid, Montreal, CANADA
PA Odyssey Pharmaceuticals, Inc., San Ramon, CA (non-U.S. corporation)
PI US 2003049688 A1 20030313
AI US 2002-154758 A1 20020524 (10)
RLI Continuation of Ser. No. US 2000-499464, filed on 7 Feb 2000, GRANTED,
Pat. No. US 6428951 Continuation of Ser. No. US 1998-17412, filed on 2
Feb 1998, GRANTED, Pat. No. US 6270964
PRAI CA 1997-2196496 19970131
DT Utility
FS APPLICATION
LN.CNT 2757
INCL INCLM: 435/007.100
INCLS: 435/007.900; 702/019.000
NCL NCLM: 435/007.100
NCLS: 435/007.900; 702/019.000
IC [7]
ICM: G01N033-53
ICS: G01N033-542; G06F019-00
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L3 ANSWER 6 OF 24 USPATFULL
AN 2002:273550 USPATFULL
TI Nucleic acids, proteins and antibodies
IN Rosen, Craig A., Laytonsville, MD, UNITED STATES
Ruben, Steven M., Olney, MD, UNITED STATES
PI US 2002151681 A1 20021017
AI US 2001-925300 A1 20010810 (9)
RLI Continuation-in-part of Ser. No. WO 2000-US5988, filed on 8 Mar 2000,
UNKNOWN
PRAI US 1999-124270P 19990312 (60)
DT Utility
FS APPLICATION
LN.CNT 29771
INCL INCLM: 530/350.000
INCLS: 536/023.500; 435/325.000; 435/320.100; 435/069.300
NCL NCLM: 530/350.000
NCLS: 536/023.500; 435/325.000; 435/320.100; 435/069.300
IC [7]
ICM: C07K014-435
ICS: C07H021-04; C12P021-02; C12N005-06
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L3 ANSWER 7 OF 24 USPATFULL
AN 2002:272900 USPATFULL
TI Stimulus-inducible protein kinase complex and methods of use therefor
IN Mercurio, Frank, San Diego, CA, UNITED STATES
Zhu, Hengyi, San Diego, CA, UNITED STATES
Barbosa, Miguel, San Diego, CA, UNITED STATES
Li, Jian Wu, San Diego, CA, UNITED STATES
Murray, Brion W., San Diego, CA, UNITED STATES
PI US 2002151021 A1 20021017
AI US 2001-844908 A1 20010427 (9)

RLI Division of Ser. No. US 1997-910820, filed on 13 Aug 1997, PATENTED
Continuation-in-part of Ser. No. US 1996-697393, filed on 26 Aug 1996,
PATENTED
DT Utility
FS APPLICATION
LN.CNT 2343
INCL INCLM: 435/194.000
INCLS: 435/325.000; 435/320.100; 435/252.300; 435/254.200; 435/348.000;
435/069.100
NCL NCLM: 435/194.000
NCLS: 435/325.000; 435/320.100; 435/252.300; 435/254.200; 435/348.000;
435/069.100
IC [7]
ICM: C12N009-12
ICS: C12P021-02; C12N005-06; C12N001-18; C12N001-21
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L3 ANSWER 8 OF 24 USPATFULL
AN 2002:258894 USPATFULL
TI 38646, a novel guanine nucleotide exchange factor and uses therefor
IN Glucksmann, Maria Alexandra, Lexington, MA, UNITED STATES
PA Millennium Pharmaceuticals, Inc., Cambridge, MA (U.S. corporation)
PI US 2002142464 A1 20021003
AI US 2001-950491 A1 20010910 (9)
PRAI US 2000-231089P 20000908 (60)
DT Utility
FS APPLICATION
LN.CNT 4625
INCL INCLM: 435/440.000
INCLS: 536/023.100
NCL NCLM: 435/440.000
NCLS: 536/023.100
IC [7]
ICM: C07H021-02
ICS: C07H021-04; C12N015-00
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L3 ANSWER 9 OF 24 USPATFULL
AN 2002:251221 USPATFULL
TI ASIP-related proteins
IN Reddy, Roopa, Sunnyvale, CA, UNITED STATES
Tang, Y. Tom, San Jose, CA, UNITED STATES
Baughn, Mariah R., San Leandro, CA, UNITED STATES
Krasnow, Randi E., Stanford, CA, UNITED STATES
PI US 2002137166 A1 20020926
AI US 2001-757781 A1 20010109 (9)
DT Utility
FS APPLICATION
LN.CNT 3608
INCL INCLM: 435/194.000
INCLS: 435/069.100; 435/325.000; 435/006.000; 435/070.210; 536/023.200
NCL NCLM: 435/194.000
NCLS: 435/069.100; 435/325.000; 435/006.000; 435/070.210; 536/023.200
IC [7]
ICM: C12Q001-68
ICS: C07H021-04; C12P021-02; C12N009-12
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L3 ANSWER 10 OF 24 USPATFULL
AN 2002:227650 USPATFULL
TI Integrin-linked kinase and its uses
IN Dedhar, Shoukat, Vancouver, CANADA
Hannigan, Greg, Toronto, CANADA
PI US 2002122801 A1 20020905

AI US 2001-840704 A1 20010423 (9)
RLI Continuation of Ser. No. US 2000-566906, filed on 9 May 2000, GRANTED,
Pat. No. US 6369205 Division of Ser. No. US 1999-390425, filed on 3 Sep
1999, GRANTED, Pat. No. US 6338958 Continuation-in-part of Ser. No. US
1997-955841, filed on 21 Oct 1997, GRANTED, Pat. No. US 6013782
Continuation-in-part of Ser. No. US 1996-752345, filed on 19 Nov 1996,
ABANDONED
PRAI US 1995-9074P 19951221 (60)
DT Utility
FS APPLICATION
LN.CNT 3236
INCL INCLM: 424/146.100
INCLS: 530/388.260; 514/044.000; 514/453.000
NCL NCLM: 424/146.100
NCLS: 530/388.260; 514/044.000; 514/453.000
IC [7]
ICM: A61K048-00
ICS: A61K039-395; A61K031-353; C07K016-40
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L3 ANSWER 11 OF 24 USPATFULL
AN 2002:72627 USPATFULL
TI Nucleic, acids, proteins, and antibodies
IN Rosen, Craig A., Laytonsville, MD, UNITED STATES
Ruben, Steven M., Olney, MD, UNITED STATES
PI US 2002039764 A1 20020404
AI US 2001-925298 A1 20010810 (9)
RLI Continuation-in-part of Ser. No. WO 2000-US5881, filed on 8 Mar 2000,
UNKNOWN
PRAI US 1999-124270P 19990312 (60)
DT Utility
FS APPLICATION
LN.CNT 20087
INCL INCLM: 435/069.100
INCLS: 435/325.000; 435/320.100; 536/023.100
NCL NCLM: 435/069.100
NCLS: 435/325.000; 435/320.100; 536/023.100
IC [7]
ICM: C12P021-02
ICS: C12N005-06; C07H021-04
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L3 ANSWER 12 OF 24 USPATFULL
AN 2002:32181 USPATFULL
TI Methods of monitoring enzyme activity
IN Griffiths, Gary, Oldham, UNITED KINGDOM
PI US 2002019002 A1 20020214
AI US 2001-877919 A1 20010607 (9)
PRAI US 2000-211313P 20000613 (60)
DT Utility
FS APPLICATION
LN.CNT 3633
INCL INCLM: 435/006.000
NCL NCLM: 435/006.000
IC [7]
ICM: C12Q001-68
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L3 ANSWER 13 OF 24 USPATFULL
AN 2002:12284 USPATFULL
TI Arrayed transfection method and uses related thereto
IN Sabatini, David M., Cambridge, MA, UNITED STATES
PI US 2002006664 A1 20020117
AI US 2001-817003 A1 20010322 (9)

PRAI US 2000-193580P 20000330 (60)
US 1999-154737P 19990917 (60)
DT Utility
FS APPLICATION
LN.CNT 2671
INCL INCLM: 435/456.000
INCLS: 435/455.000; 435/007.210
NCL NCLM: 435/456.000
NCLS: 435/455.000; 435/007.210
IC [7]
ICM: G01N033-567
ICS: C12N015-86; C12N015-85
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L3 ANSWER 14 OF 24 USPATFULL
AN 2002:75564 USPATFULL
TI Integrin-linked kinase and its uses
IN Dedhar, Shoukat, Vancouver, CANADA
Hannigan, Greg, Ontario, CANADA
PA Sunnybrook Health Science Centre, Toronto, CANADA (non-U.S. corporation)
PI US 6369205 B1 20020409
AI US 2000-566906 20000509 (9)
RLI Division of Ser. No. US 1999-390425, filed on 3 Sep 1999
Continuation-in-part of Ser. No. US 1997-955841, filed on 21 Oct 1997,
now patented, Pat. No. US 6013782 Continuation-in-part of Ser. No. US
1996-752345, filed on 19 Nov 1996, now abandoned
PRAI US 1995-9074P 19951221 (60)
DT Utility
FS GRANTED
LN.CNT 3200
INCL INCLM: 530/388.260
INCLS: 530/388.100; 530/387.100; 530/388.150; 424/139.100; 424/142.100
NCL NCLM: 530/388.260
NCLS: 424/139.100; 424/142.100; 530/387.100; 530/388.100; 530/388.150
IC [7]
ICM: C07K016-00
ICS: C12P021-08; A61K039-395
EXF 530/387.1; 530/387.2; 530/388.1; 530/388.15; 530/388.26; 424/139.1;
424/142.1
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L3 ANSWER 15 OF 24 USPATFULL
AN 2002:9751 USPATFULL
TI Integrin-linked kinase and its uses
IN Dedhar, Shoukat, Vancouver, CANADA
Hannigan, Greg, Ontario, CANADA
PA Sunnybrook Health Science Centre, Toronto, CANADA (non-U.S. corporation)
PI US 6338958 B1 20020115
AI US 1999-390425 19990903 (9)
RLI Continuation of Ser. No. US 1998-35706, filed on 5 Mar 1998, now
patented, Pat. No. US 6001622 Continuation-in-part of Ser. No. US
1997-955841, filed on 21 Oct 1997, now patented, Pat. No. US 6013782
Continuation-in-part of Ser. No. US 1996-752345, filed on 19 Nov 1996,
now abandoned
PRAI US 1995-9074P 19951221 (60)
DT Utility
FS GRANTED
LN.CNT 3203
INCL INCLM: 435/194.000
INCLS: 435/004.000; 435/015.000; 530/350.000
NCL NCLM: 435/194.000
NCLS: 435/004.000; 435/015.000; 530/350.000
IC [7]
ICM: C12N009-12

ICS: C12Q001-00; C12Q001-48; C07K001-00
EXF 530/350; 435/4; 435/7.1; 435/15; 435/194; 514/1
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L3 ANSWER 16 OF 24 USPATFULL
AN 2001:116764 USPATFULL
TI Ataxia-telangiectasia gene and its genomic organization
IN Shiloh, Yosef, Tel Aviv, Israel
PA Ramot-University Authority for Applied Research and Industrial
Development, Tel Aviv, Israel (non-U.S. corporation)
PI US 6265158 B1 20010724
WO 9636691 19961121
AI US 1998-952014 19980202 (8)
WO 1996-US7025 19960516
19980202 PCT 371 date
19980202 PCT 102(e) date
RLI Continuation-in-part of Ser. No. US 1996-629001, filed on 8 Apr 1996,
now patented, Pat. No. US 5858661 Continuation-in-part of Ser. No. US
1995-441822, filed on 16 May 1995, now patented, Pat. No. US 5756288
DT Utility
FS GRANTED
LN.CNT 3109
INCL INCLM: 435/006.000
INCLS: 536/023.100; 536/024.300; 536/024.310
NCL NCLM: 435/006.000
NCLS: 536/023.100; 536/024.300; 536/024.310
IC [7]
ICM: C12Q001-68
ICS: C07H021-04
EXF 435/6; 536/23.1; 536/24.3; 536/24.31
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L3 ANSWER 17 OF 24 USPATFULL
AN 2001:107664 USPATFULL
TI Stimulus-inducible protein kinase complex and methods of use therefor
IN Mercurio, Frank, San Diego, CA, United States
Zhu, Hengyi, San Diego, CA, United States
Barbosa, Miguel, San Diego, CA, United States
Li, Jian Wu, San Diego, CA, United States
Murray, Brion W., San Diego, CA, United States
PA Signal Pharmaceuticals, Inc., San Diego, CA, United States (U.S.
corporation)
PI US 6258579 B1 20010710
AI US 1997-910820 19970813 (8)
RLI Continuation-in-part of Ser. No. US 1996-697393, filed on 26 Aug 1996,
now patented, Pat. No. US 5972674
DT Utility
FS GRANTED
LN.CNT 1713
INCL INCLM: 435/194.000
NCL NCLM: 435/194.000
IC [7]
ICM: C12N009-12
EXF 435/194
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L3 ANSWER 18 OF 24 USPATFULL
AN 2001:48208 USPATFULL
TI Ataxia-telangiectasia gene
IN Shiloh, Yosef, Tel Aviv, Israel
Tagle, Danilo A., Gaithersburg, MD, United States
Collins, Francis, Rockville, MD, United States
PA The United States of America as represented by the Department of Health
and Human Services, Washington, DC, United States (U.S. government)

Ramot University Authority for Applied Research and Industrial Dev.,
Israel (non-U.S. corporation)

PI US 6211336 B1 20010403
WO 9636695 19961121

AI US 1998-952127 19980226 (8)
WO 1996-US7040 19960516
19980226 PCT 371 date
19980226 PCT 102(e) date

RLI Continuation-in-part of Ser. No. US 1995-508836, filed on 28 Jul 1995,
now patented, Pat. No. US 5777093 Continuation-in-part of Ser. No. US
1995-493092, filed on 21 Jun 1995, now patented, Pat. No. US 5728807
Continuation-in-part of Ser. No. US 1995-441822, filed on 16 May 1995,
now patented, Pat. No. US 5756288

DT Utility
FS Granted
LN.CNT 2279

INCL INCLM: 530/350.000
INCLS: 530/326.000

NCL NCLM: 530/350.000
NCLS: 530/326.000

IC [7]
ICM: C07K001-00
ICS: C07K014-00; C07K017-00

EXF 530/326; 530/350
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L3 ANSWER 19 OF 24 USPATFULL

AN 2001:36598 USPATFULL

TI Mutated forms of the ataxia-telangiectasia gene and method to screen for
a partial A-T phenotype

IN Shiloh, Yosef, Tel Aviv, Israel

PA Ramot-University Authority for Applied Research and Industrial
Development Ltd., Tel Aviv, Israel (non-U.S. corporation)

PI US 6200749 B1 20010313

AI US 1996-642274 19960503 (8)

RLI Continuation-in-part of Ser. No. US 1996-629001, filed on 8 Apr 1996
Continuation-in-part of Ser. No. US 1995-441822, filed on 16 May 1995,
now patented, Pat. No. US 5756288

DT Utility
FS Granted
LN.CNT 3090

INCL INCLM: 435/006.000
INCLS: 536/023.500

NCL NCLM: 435/006.000
NCLS: 536/023.500

IC [7]
ICM: C12Q001-68
ICS: C07H021-04

EXF 435/6; 435/91.2; 435/91.21; 536/23.5
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L3 ANSWER 20 OF 24 USPATFULL

AN 2000:164282 USPATFULL

TI Serine/threonine protein kinases

IN Bandman, Olga, Mountain View, CA, United States
Tang, Y. Tom, San Jose, CA, United States
Goli, Surya K., San Jose, CA, United States
Corley, Neil C., Mountain View, CA, United States
Guegler, Karl J., Menlo Park, CA, United States
Gorgone, Gina A., Boulder Creek, CA, United States
Hillman, Jennifer L., Mountain View, CA, United States

PA Incyte Pharmaceuticals, Inc., Palo Alto, CA, United States (U.S.
corporation)

PI US 6156523 20001205

AI US 1998-153939 19980916 (9)
 RLI Continuation-in-part of Ser. No. US 1996-749902, filed on 15 Nov 1996,
 now patented, Pat. No. US 5985635
 DT Utility
 FS Granted
 LN.CNT 2733
 INCL INCLM: 435/007.100
 INCLS: 424/094.100; 424/094.300; 435/069.100; 435/183.000; 435/194.000;
 530/350.000; 536/023.100; 536/023.500
 NCL NCLM: 435/007.100
 NCLS: 424/094.100; 424/094.300; 435/069.100; 435/183.000; 435/194.000;
 530/350.000; 536/023.100; 536/023.500
 IC [7]
 ICM: A61K038-43
 ICS: C12N015-52; C12N009-00; C07K014-00; G01N023-53
 EXF 424/94.1; 424/94.3; 435/69.1; 435/183; 435/194; 530/350; 536/23.1;
 536/23.5
 CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L3 ANSWER 21 OF 24 USPATFULL
 AN 1999:163477 USPATFULL
 TI Integrin-linked kinase and its use
 IN Dedhar, Shoukat, Vancouver, Canada
 Hannigan, Greg, Ontario, Canada
 PA Sunnybrook Health Science Centre, Ontario, Canada (non-U.S. corporation)
 PI US 6001622 19991214
 AI US 1998-35706 19980305 (9)
 RLI Continuation-in-part of Ser. No. US 1997-955841, filed on 21 Oct 1997
 which is a continuation-in-part of Ser. No. US 1996-752345, filed on 19
 Nov 1996
 PRAI US 1995-9074P 19951221 (60)
 DT Utility
 FS Granted
 LN.CNT 3148
 INCL INCLM: 435/194.000
 INCLS: 435/015.000
 NCL NCLM: 435/194.000
 NCLS: 435/015.000
 IC [6]
 ICM: C12N009-12
 ICS: C12Q001-48
 EXF 435/194; 435/15
 CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L3 ANSWER 22 OF 24 USPATFULL
 AN 1999:146335 USPATFULL
 TI Nucleic acids encoding novel human serine/threonine protein kinases
 IN Bandman, Olga, Mountain View, CA, United States
 Goli, Surya K., Sunnyvale, CA, United States
 Hillman, Jennifer L., San Jose, CA, United States
 PA Incyte Pharmaceuticals, Inc., Palo Alto, CA, United States (U.S.
 corporation)
 PI US 5985635 19991116
 AI US 1996-749902 19961115 (8)
 DT Utility
 FS Granted
 LN.CNT 2520
 INCL INCLM: 435/194.000
 INCLS: 435/069.100; 435/252.300; 435/254.110; 435/320.100; 435/325.000;
 536/023.200; 536/024.310
 NCL NCLM: 435/194.000
 NCLS: 435/069.100; 435/252.300; 435/254.110; 435/320.100; 435/325.000;
 536/023.200; 536/024.310
 IC [6]

ICM: C12N015-54
ICS: C12N009-12
EXF 435/69.1; 435/325; 435/254.11; 435/252.3; 435/194; 435/320.1; 536/23.2;
536/24.31
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L3 ANSWER 23 OF 24 USPATFULL
AN 1999:132563 USPATFULL
TI Stimulus-inducible protein kinase complex and methods of use therefor
IN Mercurio, Frank, San Diego, CA, United States
Zhu, Hengyi, San Diego, CA, United States
Barbosa, Miguel, San Diego, CA, United States
PA Signal Pharmaceuticals, Inc., San Diego, CA, United States (U.S.
corporation)
PI US 5972674 19991026
AI US 1996-697393 19960826 (8)
DT Utility
FS Granted
LN.CNT 945
INCL INCLM: 435/194.000
INCLS: 530/350.000; 530/352.000
NCL NCLM: 435/194.000
NCLS: 530/350.000; 530/352.000
IC [6]
ICM: C12N009-12
EXF 530/350; 530/352; 435/194
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L3 ANSWER 24 OF 24 USPATFULL
AN 1999:4329 USPATFULL
TI Ataxia-telangiectasia gene and its genomic organization
IN Shiloh, Yosef, Tel Aviv, Israel
PA RAMOT-University Authority for Applied Research and Industrial
Development, Tel Aviv, Israel (non-U.S. corporation)
PI US 5858661 19990112
AI US 1996-629001 19960408 (8)
RLI Continuation-in-part of Ser. No. US 1995-441822, filed on 16 May 1995,
now patented, Pat. No. US 5756288
DT Utility
FS Granted
LN.CNT 3461
INCL INCLM: 435/006.000
INCLS: 536/023.500; 935/077.000; 935/078.000
NCL NCLM: 435/006.000
NCLS: 536/023.500
IC [6]
ICM: C12Q001-68
ICS: C07H021-04
EXF 436/6; 436/91.2; 436/975; 536/23.5; 536/22.1; 536/24.3; 536/24.33
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

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L3 ANSWER 1 OF 24 USPATFULL
ACCESSION NUMBER: 2003:114491 USPATFULL
TITLE: Assay for measuring enzyme activity in vivo
INVENTOR(S): Craig, Roger Kingdon, Smallwood, UNITED KINGDOM
Green, Simon, Invergowrie, UNITED KINGDOM
Colyer, John, Bardsey, UNITED KINGDOM

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003079235	A1	20030424

APPLICATION INFO.: US 2002-147354 A1 20020516 (10)
RELATED APPLN. INFO.: Continuation-in-part of Ser. No. WO 2000-GB4348, filed
on 15 Nov 2000, UNKNOWN

	NUMBER	DATE
PRIORITY INFORMATION:	GB 1999-27331	19991118
DOCUMENT TYPE:	Utility	
FILE SEGMENT:	APPLICATION	
LEGAL REPRESENTATIVE:	FROMMER LAWRENCE & HAUG, 745 FIFTH AVENUE- 10TH FL., NEW YORK, NY, 10151	
NUMBER OF CLAIMS:	22	
EXEMPLARY CLAIM:	1	
NUMBER OF DRAWINGS:	1 Drawing Page(s)	
LINE COUNT:	2708	
SUMM	[0141] Further examples of protein kinases identified to date include the protein tyrosine kinase subfamily (such as PDGF receptors, EGF receptors, src family kinases (see Brown and Cooper, 1996, Biochimica and Biophysica Acta 1287: 121-149 for a review), the JAK kinase family (such as JAK1, JAK2 and tyk2), Erb B2, Bcr-Abl, Alk, Trk, Res/Sky--for a detailed review see Al-Obeidi et al., 1998, Biopolymers (Peptide Science), Vol 47: 197-223), the MAP kinase pathway subfamily (such as the MAP family, the ERK family, the MEK family, the MEKK family, RAF-1 and JNK), the cyclin-dependent kinase subfamily (such as p34.sup.cdc2 and cdk2--see Nigg, 1995, Bioessays 17: 471-480 for a review), Weel/Myt1, polo-like kinases (such as plk1, Plx1, POLO, Snk, Fnk/Prk Sak-a, Sak-b--see Lane and Nigg, 1997, Trends in Cell Biol. 7: 63-68), the receptor serine kinase subfamily, protein kinase C (PK-C), cyclic-AMP dependent kinase (PK-A), cyclic-GMP dependent kinase , Ca2+/calmodulin dependent kinases (such as CaM kinase I, II and IV), DNA dependent protein kinase), phosphoinositide 3-kinases, PDK-1, the p21-activated protein kinase family (PAKs), such as Pak1, Pak2 and Pak3--see Sells and Chemoff, 1997, Trends in Cell Biol. 7: 162-167), p70 S6 kinase , Ikb kinase , casein kinase II, glycogen-synthase kinases.	
SUMM	. . . protozoal antigens and parasitic antigens. Candidate modulators additionally comprise proteins, lipoproteins, glycoproteins, phosphoproteins and nucleic acids (e.g., RNAs such as ribozymes or antisense nucleic acids). Proteins or polypeptides which can be screened using the methods of the present invention include hormones,.	
SUMM	. . . described above, but may eliminate or encode products which eliminate deleterious proteins. Such nucleic acid sequences are antisense RNA and ribozymes , as well as DNA expression constructs that encode them. Note that antisense RNA molecules, ribozymes or genes encoding them may be administered to a test cell or organism by a method of nucleic acid delivery that is known in the art, as described below. Inactivating nucleic acid sequences may encode a ribozyme or antisense RNA specific for the a target mRNA. Ribozymes of the hammerhead class are the smallest known, and tend themselves both to in vitro production and delivery to cells.	
SUMM	[0269] When the end product (e.g. an antisense RNA molecule or ribozyme) is administered directly, the dosage to be administered is directly proportional to the amount needed per cell and the number. . .	

L3 ANSWER 2 OF 24 USPATFULL

ACCESSION NUMBER: 2003:100088 USPATFULL
TITLE: Treatment methods based on microcompetition for a limiting GABP complex
INVENTOR(S): Polansky, Hanan, Rochester, NY, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003069199	A1	20030410
APPLICATION INFO.:	US 2002-219334	A1	20020815 (10)
RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 2000-732360, filed on 7 Dec 2000, PENDING		
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	APPLICATION		
LEGAL REPRESENTATIVE:	Hanan Polansky, 3159 S. Winton Rd., Rochester, NY, 14623		
NUMBER OF CLAIMS:	26		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	28 Drawing Page(s)		
LINE COUNT:	14837		

DETD [0543] Another aspect of the invention pertains to administration of a polynucleotide as antisense/antigene, **ribozyme**, triple helix, homologous nucleic acids, peptide nucleic acids, or microcompetitions, equivalent polynucleotides, or homologous polynucleotides, isolated from, or substantially free. . . .

DETD [0544] The following sections present standard protocols for the formulation of such polynucleotides. Since antisense/antigene, **ribozyme**, triple helix, homologous nucleic acids, peptide nucleic acids, and microcompetition agents are nucleic acid based, they share protocols for their. . . .

DETD [0570] (b) **Ribozymes**

DETD . . . transcription or translation of the target gene, or by inducing enzyme-mediated transcript degradation by RNase H or a similar enzyme, **ribozymes** offer an alternative approach. **Ribozymes** are RNA molecules which natively bind to and cleave target transcripts. Typical **ribozymes** bind to and cleave RNA at specific sites, however hammerhead **ribozymes** cleave target transcripts at sites directed by flanking nucleotide sequences which bind to the target site. The use of hammerhead **ribozymes** is preferred because the only sequence requirement for their activity is the UG dinucleotide arranged in the 5'-3' orientation. Hammerhead. . . . in the art (see, for example Doherty 2001.sup.138, or Goodchild 2000.sup.139). In a preferred embodiment, the sequence targeted by the **ribozyme** lies near the 5' end of the transcript. That will result cleavage of the transcript near the translation initiation site. . . .

DETD [0572] **Ribozymes** identified in Tetrahymena thermophila, which employ an eight base pair active site which duplexes with the target RNA molecule, are included in this invention. This invention includes those **ribozymes**, described and characterized by Cech and coworkers (i.e. IVS or L-19IVS RNA), which target eight base-pair sequences in a gene. . . . For the catalytic sequence of these agents see, for instance, U.S. Pat. No. 5,093,246, incorporated entirely herein by reference. Any **ribozyme** or hammerhead **ribozyme** molecules that target RNA sequences expressed by a foreign polynucleotide, disrupted gene or gene in a disrupted pathway, are included. . . .

DETD [0573] **Ribozymes**, being RNA molecules of specific sequence, may be synthesized with modified nucleotides which enable better targeting to the host cell. . . . cleave and disrupt transcripts of foreign DNA or disrupted genes or genes in a disrupting pathway. The catalytic nature of **ribozymes** permits their effective use at concentrations below those needed for traditional antisense agents.

DETD [0574] Identification of **ribozyme** cleavage sites within a transcript of interest is accomplished with any of a number of computer algorithms which scan linear. . . . well known in the art, for their potential to form secondary structures which may interfere with the action of targeted **ribozyme** agents. Alternatively, empirical assays employing ribonucleases may be used to probe the accessibility of identified target sequences.

DETD [0575] **Ribozymes** comprise a unique class of oligonucleotides which bind to specific ribonucleic acid targets and promote their hydrolysis. The design of **ribozyme** agents is well known to those skilled in the art. In order to prepare effective **ribozyme** agents, initially a suitable target sequence must be identified which confers specificity to the agent in order to minimize unwanted side effects and maximize efficacy. Once that target is identified the **ribozyme** agent is synthesized using standard oligonucleotide synthesis procedures such as those exemplified herein. Delivery to the target cell may be.

DETD [0576] Ensuring the purity and efficacy of **ribozyme** agents may be more important than for other nucleic acid agents because their intended effects, namely the hydrolysis of target. . . . extensive preclinical testing is essential to minimize unwanted side effects. These risks are, however, outweighed by the potential effectiveness of **ribozyme** agents.

DETD . . . purines within the target sequence and vice versa, which inhibit transcription of the target sequence. The effectiveness of a targeted **triplex** forming oligonucleotide may be enhanced by including a "switchback" motif composed of alternating 5'-3' and 3'-5' regions of purines and.

DETD [0580] **Triples** agent formulation begins with selection of an appropriate target sequence within the cells to be treated. That target may be. . . the target is double stranded DNA, the most effective targets surround and include the transcriptional regulatory regions. Formation of a **triplex** between the agent and the target will inhibit the binding of RNA polymerase or other requisite transcriptional regulatory factors which.

DETD [0581] **Triples** agents may be synthesized to be more resistant to cellular and extracellular nucleases by the inclusion of modified nucleotides such. . . of the base intercalating agent acridine, may be incorporated into the therapeutic agent to restore desirable binding properties to the **triplex** forming oligonucleotide. Alternatively, if the intracellular target is an mRNA, C-5 propyne pyrimidines may be included in the synthetic oligophosphorothioate.

DETD [0582] The affinity of **triplex** agents for their respective targets may be assessed by electrophoretic gel retardation assays. The formation of **triplex** structures will retard migration through an electrophoretic gel. Similarly, UV melting experiments can assess the stability of any **triplex** agent binding to its target. In these assays **triplex** agents are mixed with their intended target in vitro and the resulting triplexes are heated (with, for example, a Haake cryothermostat) while monitoring their UV absorbance (with, for example, a Kontron-Uvikon 940 spectrophotometer) (on design of **triplex** forming oligonucleotides see, for instance, Francois (1999.sup.140)).

DETD [0583] **Triples** forming agents are simply oligonucleotides designed to form triple helices with the target intracellular nucleic acid. Accordingly, their synthesis, purification.

DETD . . . isolated, amplified, if necessary, digested with one or several restriction endonucleases, and the fragments separated by gel electrophoresis. Sequence specific **ribozymes** are then used to detect specific mutations by development or loss of a **ribozyme** cleavage site.

DETD [1117] HRG activated the MAP **kinase** isoforms p44ERK1 and p42ERK2 and the **p70/p85 S6 kinase** in AU565, T47D and HC11 cells. HRG stimulation caused growth arrest of the AU565 cells and proliferation of the T47D or HC11 cells. HRG also stimulated tyrosine phosphorylation and in vitro **kinase** activity of ErbB-2. When TPA, another ERK agent, activated PKC HRG was no longer able to activate ErbB-2 in T47D cells, blocking cell proliferation. Activation of ErbB-2 by point mutation or monoclonal antibodies also stimulated MAPK and **p70/p85 S6 kinase** pathways. The same monoclonal antibodies

also induced AU565 cell differentiation (Marte 1995, *ibid*).

DETD [2207] .sup.90 Marte B M, Graus-Porta D, Jeschke M, Fabbro D, Hynes N E, Taverna D. NDF/hereregulin activates MAP **kinase** and **p70/p85 S6 kinase** during proliferation or differentiation of mammary epithelial cells. *Oncogene* 1995 Jan 5;10(1):167-75.

DETD [2255] .sup.138 Doherty, E. A and Doudna. **Ribozyme** structures and mechanisms. *J.A. Ann. Rev. Biophys. Biomol Struct.* 30, 457-475, 2001.

DETD [2256] .sup.139 Goodchild, J. Hammerhead **ribozymes**: biochemical and chemical considerations. *Curr. Opin. Mol. Ther* 2, 272-281, 2000. Review.

DETD [2257] .sup.140 Francois, J.-C., Lacoste, J., Lacroix, L. and J.-L. Mergny. Design of Antisense and **Triplex**-Forming Oligonucleotides. In *Methods in Enzymology*, ed. M Ian Phillips, v313, 1999, Academic Press, pp74-95

L3 ANSWER 3 OF 24 USPATFULL

ACCESSION NUMBER: 2003:99511 USPATFULL
 TITLE: Drug discovery assays based on microcompetition for a limiting GABP complex
 INVENTOR(S): Polansky, Hanan, Rochester, NY, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003068616	A1	20030410
APPLICATION INFO.:	US 2002-223050	A1	20020814 (10)
RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 2000-732360, filed on 7 Dec 2000, PENDING		
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	APPLICATION		
LEGAL REPRESENTATIVE:	Hanan Polansky, 3159 S. Winton Rd., Rochester, NY, 14623		
NUMBER OF CLAIMS:	55		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	28 Drawing Page(s)		
LINE COUNT:	14981		

DETD [0517] Another aspect of the invention pertains to administration of a polynucleotide as antisense/antigene, **ribozyme**, triple helix, homologous nucleic acids, peptide nucleic acids, or microcompetitors, equivalent polynucleotides, or homologous polynucleotides, isolated from, or substantially free. . .

DETD [0518] The following sections present standard protocols for the formulation of such polynucleotides. Since antisense/antigene, **ribozyme**, triple helix, homologous nucleic acids, peptide nucleic acids, and microcompetition agents are nucleic acid based, they share protocols for their. . .

DETD [0543] (b) **Ribozymes**

DETD . . . transcription or translation of the target gene, or by inducing enzyme-mediated transcript degradation by RNase H or a similar enzyme, **ribozymes** offer an alternative approach. **Ribozymes** are RNA molecules which natively bind to and cleave target transcripts. Typical **ribozymes** bind to and cleave RNA at specific sites, however hammerhead **ribozymes** cleave target transcripts at sites directed by flanking nucleotide sequences which bind to the target site. The use of hammerhead **ribozymes** is preferred because the only sequence requirement for their activity is the UG dinucleotide arranged in the 5'-3' orientation. Hammerhead. . . in the art (see, for example Doherty 2001.sup.138, or Goodchild 2000.sup.139). In a preferred embodiment, the sequence targeted by the **ribozyme** lies near the 5' end of the transcript. That will result cleavage of the transcript near the translation initiation site. . .

DETD [0545] **Ribozymes** identified in *Tetrahymena thermophila*, which employ an eight base pair active site which duplexes with the target RNA

molecule, are included in this invention. This invention includes those **ribozymes**, described and characterized by Cech and coworkers (i.e. IVS or L-191VS RNA), which target eight base-pair sequences in a gene. . . . For the catalytic sequence of these agents see, for instance, U.S. Pat. No. 5,093,246, incorporated entirely herein by reference. Any **ribozyme** or hammerhead **ribozyme** molecules that target RNA sequences expressed by a foreign polynucleotide, disrupted gene or gene in a disrupted pathway, are included. . . .

DETD [0546] **Ribozymes**, being RNA molecules of specific sequence, may be synthesized with modified nucleotides which enable better targeting to the host cell. . . . cleave and disrupt transcripts of foreign DNA or disrupted genes or genes in a disrupting pathway. The catalytic nature of **ribozymes** permits their effective use at concentrations below those needed for traditional antisense agents.

DETD [0547] Identification of **ribozyme** cleavage sites within a transcript of interest is accomplished with any of a number of computer algorithms which scan linear. . . . well known in the art, for their potential to form secondary structures which may interfere with the action of targeted **ribozyme** agents. Alternatively, empirical assays employing ribonucleases may be used to probe the accessibility of identified target sequences.

DETD [0548] **Ribozymes** comprise a unique class of oligonucleotides which bind to specific ribonucleic acid targets and promote their hydrolysis. The design of **ribozyme** agents is well known to those skilled in the art. In order to prepare effective **ribozyme** agents, initially a suitable target sequence must be identified which confers specificity to the agent in order to minimize unwanted side effects and maximize efficacy. Once that target is identified the **ribozyme** agent is synthesized using standard oligonucleotide synthesis procedures such as those exemplified herein. Delivery to the target cell may be. . . .

DETD [0549] Ensuring the purity and efficacy of **ribozyme** agents may be more important than for other nucleic acid agents because their intended effects, namely the hydrolysis of target. . . . extensive preclinical testing is essential to minimize unwanted side effects. These risks are, however, outweighed by the potential effectiveness of **ribozyme** agents.

DETD . . . purines within the target sequence and vice versa, which inhibit transcription of the target sequence. The effectiveness of a targeted **triplex** forming oligonucleotide may be enhanced by including a "switchback" motif composed of alternating 5'-3' and 3'-5' regions of purines and. . . .

DETD [0553] **Triples** agent formulation begins with selection of an appropriate target sequence within the cells to be treated. That target may be. . . . the target is double stranded DNA, the most effective targets surround and include the transcriptional regulatory regions. Formation of a **triplex** between the agent and the target will inhibit the binding of RNA polymerase or other requisite transcriptional regulatory factors which. . . .

DETD [0554] **Triples** agents may be synthesized to be more resistant to cellular and extracellular nucleases by the inclusion of modified nucleotides such. . . . of the base intercalating agent acridine, may be incorporated into the therapeutic agent to restore desirable binding properties to the **triplex** forming oligonucleotide. Alternatively, if the intracellular target is an mRNA, C-5 propyne pyrimidines may be included in the synthetic oligophosphorothioate. . . .

DETD [0555] The affinity of **triplex** agents for their respective targets may be assessed by electrophoretic gel retardation assays. The formation of **triplex** structures will retard migration through an electrophoretic gel. Similarly, UV melting experiments can assess the stability of any **triplex** agent binding to its target. In these assays **triplex** agents are mixed with their intended target in

vitro and the resulting triplexes are heated (with, for example, a Haake cryothermostat) while monitoring their UV absorbance (with, for example, a Kontron-Uvikon 940 spectrophotometer) (on design of **triplex** forming oligonucleotides see, for instance, Francois (1999.sup.140)).

DETD [0556] **Trip**lex forming agents are simply oligonucleotides designed to form triple helices with the target intracellular nucleic acid. Accordingly, their synthesis, purification. . .

DETD . . . isolated, amplified, if necessary, digested with one or several restriction endonucleases, and the fragments separated by gel electrophoresis. Sequence specific **ribozymes** are then used to detect specific mutations by development or loss of a **ribozyme** cleavage site.

DETD [1089] HRG activated the MAP **kinase** isoforms p44ERK1 and p42ERK2 and the **p70/p85 S6 kinase** in AU565, T47D and HC11 cells. HRG stimulation caused growth arrest of the AU565 cells and proliferation of the T47D or HC11 cells. HRG also stimulated tyrosine phosphorylation and in vitro **kinase** activity of ErbB-2. When TPA, another ERK agent, activated PKC HRG was no longer able to activate ErbB-2 in T47D cells, blocking cell proliferation. Activation of ErbB-2 by point mutation or monoclonal antibodies also stimulated MAPK and **p70/p85 S6 kinase** pathways. The same monoclonal antibodies also induced AU565 cell differentiation (Marte 1995, *ibid*).

DETD [2187] .sup.90 Marte B M, Graus-Porta D, Jeschke M, Fabbro D, Hynes N E, Taverna D. NDF/heregulin activates MAP **kinase** and **p70/p85 S6 kinase** during proliferation or differentiation of mammary epithelial cells. *Oncogene* Jan. 5, 1995;10(1):167-75.

DETD [2235] .sup.138 Doherty, E. A and Doudna. **Ribozyme** structures and mechanisms. *J. A. Ann. Rev. Biophys. Biomol Struct.* 30, 457-475, 2001.

DETD [2236] .sup.139 Goodchild, J. Hammerhead **ribozymes**: biochemical and chemical considerations. *Curr. Opin. Mol. Ther* 2, 272-281, 2000. Review.

DETD [2237] .sup.140 Francois, J. -C., Lacoste, J., Lacroix, L. and J.-L. Mergny. Design of Antisense and **Trip**lex-Forming Oligonucleotides. In *Methods in Enzymology*, ed. M Ian Phillips, v313, 1999, Academic Press, pp74-95

L3 ANSWER 4 OF 24 USPATFULL

ACCESSION NUMBER: 2003:78501 USPATFULL
 TITLE: Nucleic acids, proteins, and antibodies
 INVENTOR(S): Rosen, Craig A., Laytonsville, MD, UNITED STATES
 Ruben, Steven M., Olney, MD, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003054421	A1	20030320
APPLICATION INFO.:	US 2002-102806	A1	20020322 (10)
RELATED APPLN. INFO.:	Continuation of Ser. No. US 2001-925298, filed on 10 Aug 2001, PENDING Continuation-in-part of Ser. No. WO 2000-US5881, filed on 8 Mar 2000, UNKNOWN		

	NUMBER	DATE
PRIORITY INFORMATION:	US 1999-124270P	19990312 (60)
DOCUMENT TYPE:	Utility	
FILE SEGMENT:	APPLICATION	
LEGAL REPRESENTATIVE:	HUMAN GENOME SCIENCES INC, 9410 KEY WEST AVENUE, ROCKVILLE, MD, 20850	
NUMBER OF CLAIMS:	24	
EXEMPLARY CLAIM:	1	
LINE COUNT:	20141	
CAS INDEXING IS AVAILABLE FOR THIS PATENT.		

SUMM . . . 90 90 HHESJ29
[Homo sapiens] >sp|G4100632|G4100632
LYMPHOID PHOSPHATASE LYP1. Length =
808

189 792931
1 576 HEGAW71

190 792943 myosin heavy chain **kinase** B [Dictyostelium
gi|1903458 3 1247 43 68 HDPRZ79
discoideum] >sp|P90648|KMH_B_DICDI
MYOSIN HEAVY CHAIN **KINASE** B (EC
2.7.1.129) (MHCK B). Length = 732

191 793104
107 250 HKGAJ80

192 793445 desmoyokin - human (fragments)
pir|A45259|A45259 1 723. . . >gi|337762
prosaposin [Homo sapiens]
>gi|337756 sphingolipid activator precursor
[Homo sapiens] Length = 524

227 813262
1 345 HFKCA89

228 815637 (AC004003) serine/threonine **kinase** RICK;
gi|3264574 3 461 92 92 HNHD566
match to protein AF027706 (PID:g3123887)
and mRNA AF027706 (NID:g3123886)
[Homo sapiens] >gi|3290172 (AF064824)
CARD-containing ICE associated **kinase**
[Homo sapiens] >gi|3342910 (AF078530)
receptor interacting prote

229 815853 calcyphosine [Homo sapiens] >gi|3075376
gnl|PID|e245872 8 667 100 100 HLHAY85
(AC004602) CAYP_HUMAN; . . . CALMODULIN.
Length = 149

241 827732 alternate name ygiG; ORF_f123 [Escherichia
gi|882580 181 282 91 95 HBGDE81
coli] >gi|1789438 (AE000387) putative **kinase**
[Escherichia coli] >pir|H65093|H65093 ygiG
protein - Escherichia coli (strain K-12)
>sp|P31055|FOLB_ECOLI PROBABLE
DIHYDRONEOPTERIN ALDOLASE (EC
4.1.2.25) (DHNA). {SUB

242 827735
541 708 HHEDU22

243 827740
716 838 HBNAP17

244 827808
86 1657 HMELR44

245 828251 (AB016869) **p70** ribosomal 56 **kinase**
beta gnl|PID|d1035383 134 949 91 91
HNGOL64
[Homo sapiens] >sp|D1035383|D1035383 **P70**
RIBOSOMAL **S6 KINASE** BETA. Length =
495

246 828357
1 768 HKIYP61

247 828449
1 723 HBXCZ22

248 828612 syntaxin 5 [Homo sapiens]
gi|886071 68 460. . .

SUMM . . . targeted cell. In another example, the invention provides a
method for delivering a single stranded nucleic acid (e.g., antisense or
ribozymes) or double stranded nucleic acid (e.g., DNA that can
integrate into the cell's genome or replicate episomally and that can.

SUMM . . . cell). In another example, the invention provides a method for

delivering a single - stranded nucleic acid (e.g., antisense or **ribozymes**) or double stranded nucleic acid (e.g., DNA that can integrate into the cell's genome or replicate episomally and that can.

SUMM [0489] Antagonists of the invention include, for example, binding and/or inhibitory antibodies, antisense nucleic acids, **ribozymes** or soluble forms of the polypeptides of the present invention (e.g., Fc fusion protein; see, e.g., Example 9). Agonists of.

SUMM . . . the-targeted cell. In another example, the invention provides a method for delivering a single stranded nucleic acid (e.g., antisense or **ribozymes**) or double stranded nucleic acid (e.g., DNA that can integrate into the cell's genome or replicate episomally and that can.

SUMM Antisense And **Ribozymes** (Antagonists)

SUMM . . . the case of double stranded antisense nucleic acids, a single strand of the duplex DNA may thus be tested, or **triplex** formation may be assayed. The ability to hybridize will depend on both the degree of complementarity and the length of. . . hybridizing nucleic acid, the more base mismatches with a RNA it may contain and still form a stable duplex (or **triplex** as the case may be). One skilled in the art can ascertain a tolerable degree of mismatch by use of.

SUMM [0710] Potential antagonists according to the invention also include catalytic RNA, or a **ribozyme** (See, e.g., PCT International Publication WO 90/11364, published Oct. 4, 1990; Sarver et al, Science 247:1222-1225 (1990). While **ribozymes** that cleave mRNA at site specific recognition sequences can be used to destroy mRNAs, the use of hammerhead **ribozymes** is preferred. Hammerhead **ribozymes** cleave mRNAs at locations dictated by flanking regions that form complementary base pairs with the target mRNA. The sole requirement is that the target mRNA have the following sequence of two bases: 5'-UG-3'. The construction and production of hammerhead **ribozymes** is well known in the art and is described more fully in Haseloff and Gerlach, Nature 334:585-591 (1988). There are numerous potential hammerhead **ribozyme** cleavage sites within the nucleotide sequence of SEQ ID NO:X. Preferably, the **ribozyme** is engineered so that the cleavage recognition site is located near the 5' end of the mRNA; i.e., to increase.

SUMM [0711] As in the antisense approach, the **ribozymes** of the invention can be composed of modified oligonucleotides (e.g. for improved stability, targeting, etc.) and should be delivered to cells which express in vivo. DNA constructs encoding the **ribozyme** may be introduced into the cell in the same manner as described above for the introduction of antisense encoding DNA. A preferred method of delivery involves using a DNA construct "encoding" the **ribozyme** under the control of a strong constitutive promoter, such as, for example, pol III, or pol II promoter, so that transfected cells will produce sufficient quantities of the **ribozyme** to destroy endogenous messages and inhibit translation. Since **ribozymes** unlike antisense molecules, are catalytic, a lower intracellular concentration is required for efficiency.

SUMM . . . by administering to a patient (a) an antisense molecule directed to the polynucleotide of the present invention, and/or (b) a **ribozyme** directed to the polynucleotide of the present invention.

L3 ANSWER 5 OF 24 USPATFULL

ACCESSION NUMBER: 2003:71403 USPATFULL

TITLE: Protein fragment complementation assays for the detection of biological or drug interactions

INVENTOR(S): Michnick, Stephen William Watson, Westmount, CANADA
Pelletier, Joelle Nina, Westmount, CANADA
Remy, Ingrid, Montreal, CANADA

PATENT ASSIGNEE(S): Odyssey Pharmaceuticals, Inc., San Ramon, CA (non-U.S.

corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003049688	A1	20030313
APPLICATION INFO.:	US 2002-154758	A1	20020524 (10)
RELATED APPLN. INFO.:	Continuation of Ser. No. US 2000-499464, filed on 7 Feb 2000, GRANTED, Pat. No. US 6428951 Continuation of Ser. No. US 1998-17412, filed on 2 Feb 1998, GRANTED, Pat. No. US 6270964		

	NUMBER	DATE
PRIORITY INFORMATION:	CA 1997-2196496	19970131
DOCUMENT TYPE:	Utility	
FILE SEGMENT:	APPLICATION	
LEGAL REPRESENTATIVE:	Isaac A. Angres, Suite 301, 2001 Jefferson Davis Highway, Arlington, VA, 22202	
NUMBER OF CLAIMS:	58	
EXEMPLARY CLAIM:	1	
NUMBER OF DRAWINGS:	7 Drawing Page(s)	
LINE COUNT:	2757	
CAS INDEXING IS AVAILABLE FOR THIS PATENT.		

DETD [0166] One of the earliest detectable events in growth factor-activated cell proliferation is the serine phosphorylation of the **S6** protein of the 40S ribosomal subunit. The discovery of serine/threonine kinases that specifically phosphorylate **S6** have considerably aided in identifying novel mitogen mediated signal transduction pathways. The serine/threonine **kinase** p70S6k has been identified as a specific **S6** phosphorylase.sup.131-136. p70S6k is activated by serine and threonine phosphorylation at specific sites in response to several mitogenic signals including serum. . . . and by mitogens such as phorbol esters. Considerable effort has been made over the last five years to determine how **p70/p85S6k** are activated in response to mitogens. Two receptor-mediated pathways have been implicated in p70S6k activation, one associated with the phosphatidylinositol-3-**kinase** (PI(3)k) and the other with the PI(3)k homologue mTOR.sup.137-144. Key to understanding of this proposal, is the fact that the. . . .

DETD . . . P., Balasubramanyam, A., Coffey, P. J., Price, D. J., Avruch, J. & Woodgett, J. R.:Cloning and expression of two human **p70 S6 kinase** polypeptides differing only at their amino termini. Mol. Cell. Biol. 11, 5541-50 (1991).

DETD [0350] 135. Reinhard, C., Thomas, G. & Kozma, S. C.:A single gene encodes two isoforms of the **p70 S6 kinase**: activation upon mitogenic stimulation. Proc. Natl. Acad. Sci. USA 89, 4052-6 (1992).

DETD . . . J., Chung, J., Fiorentino, D. F., Flanagan, W. M., Blenis, J. & Crabtree, G. R.:Rapamycin selectively inhibits interleukin-2 activation of **p70 S6 kinase**. Nature 358, 70-3 (1992).

DETD . . . & Gelfand, E. W.:Failure of rapamycin to block proliferation once resting cells have entered the cell cycle despite inactivation of **p70 S6 kinase**. J. Biol. Chem. 268,12062-8 (1993).

DETD [0356] 141. Calvo, V., Crews, C. M., Vik, T. A. & Bierer, B. E.:Interleukin 2 stimulation of **p70 S6 kinase** activity is inhibited by the immunosuppressant rapamycin. Proc. Natl. Acad. Sci. USA 89, 7571-5 (1992).

DETD . . . P., Andrabi, K., Kozlowski, M. T., Grove, J. R. & Avruch, J.:Multiple independent inputs are required for activation of the **p70 S6 kinase**. Mol. Cell. Biol. 15, 2333-40 (1995).

DETD . . . Burgering, B. M., Wennstrom, S., Claesson-Welsh, L., Heldin, C. H., Bos, J. L., Kozma, S. C. & Thomas, G.:Activation of **p70/**

p85 S6 kinase by a pathway independent of
 p21 ras [see comments]. Nature 371, 426-9 (1994).
 DETD [0359] 144. Cheatham, L., Monfar, M., Chou, M. M. & Blenis,
 J.:Structural and functional analysis of **pp70S6k**. Proc. Natl.
 Acad. Sci. USA 92,11696-700 (1995).
 CLM What is claimed is:
 10. The method of claim 2 wherein said detected molecule is a nucleic
 acid or a **ribozyme**.

L3 ANSWER 6 OF 24 USPATFULL

ACCESSION NUMBER: 2002:273550 USPATFULL
 TITLE: Nucleic acids, proteins and antibodies
 INVENTOR(S): Rosen, Craig A., Laytonsville, MD, UNITED STATES
 Ruben, Steven M., Olney, MD, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002151681	A1	20021017
APPLICATION INFO.:	US 2001-925300	A1	20010810 (9)
RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. WO 2000-US5988, filed on 8 Mar 2000, UNKNOWN		

	NUMBER	DATE
PRIORITY INFORMATION:	US 1999-124270P	19990312 (60)
DOCUMENT TYPE:	Utility	
FILE SEGMENT:	APPLICATION	
LEGAL REPRESENTATIVE:	HUMAN GENOME SCIENCES INC, 9410 KEY WEST AVENUE, ROCKVILLE, MD, 20850	
NUMBER OF CLAIMS:	23	
EXEMPLARY CLAIM:	1	
LINE COUNT:	29771	
CAS INDEXING IS AVAILABLE FOR THIS PATENT.		
SUMM	2 988 94 94	HL4AF38

>pir|S34130|S34130 serine/threonine-specific
 protein **kinase** PLK (EC 2.7.1.-)-human
 >sp|P53350|PLK1 HUMAN
 SERINE/THREONINE-PROTEIN **KINASE**
 PLK (EC 2.7.1.-) (PLK-1) (SERINE-
 THREONINE PROTEIN **KINASE** 13)
 (STPK 13). Length = 603

329	829196				
	1	432		HL1AR10	
330	829197			TAK1 binding protein [Homo sapiens]	
	gi 1401126		1	252	75. . . 322
	HJMBB19				
364	829285				
	706	912		HKADQ69	
365	829287				
	134	358		HJAAB29	
366	829295				
	81	212		HJACK32	
367	829296				
	352	666		HISAN67	
368	829297			mitotic kinase -like protein-1 [Homo sapiens]	
	gi 34672		1	225	98 98 HJPBA19
				>pir S28262 S28262 kinesin-related protein	
				MKLP-1 -human >sp Q02241 MKLP HUMAN	
				MITOTIC KINESIN-LIKE PROTEIN-1. Length =	
. . .	88			HLWBS80	
				PININ. Length = 773	
417	831355			GTP-binding protein-mouse Length = 198	
	pir S39543 S39543		128	730	99 100 HKMAB33

418 831420 (AB016869) **p70** ribosomal **S6**
kinase beta gnl|PID|d10353831 672 91
92 HWBAS06
[Homo sapiens]>sp|D1035383|D1035383 **P70**
RIBOSOMAL S6 KINASE BETA. Length =
495

419 831702 Gem [Homo sapiens]>pir|A54575|A54575 35K
gi|544493 100 1107 93 93 H2LAD84
GTP-binding protein Gem-human

SUMM . . . targeted cell. In another example, the invention provides a method for delivering a single stranded nucleic acid (e.g., antisense or **ribozymes**) or double stranded nucleic acid (e.g., DNA that can integrate into the cell's genome or replicate episomally and that can.

SUMM . . . cancer cell). In another example, the invention provides a method for delivering a single stranded nucleic acid (e.g., antisense or **ribozymes**) or double stranded nucleic acid (e.g., DNA that can integrate into the cell's genome or replicate episomally and that can.

SUMM [0507] Antagonists of the invention include, for example, binding and/or inhibitory antibodies, antisense nucleic acids, **ribozymes** or soluble forms of the polypeptides of the present invention (e.g., Fc fusion protein; see, e.g., Example 9). Agonists of. . .

SUMM . . . targeted cell. In another example, the invention provides a method for delivering a single stranded nucleic acid (e.g., antisense or **ribozymes**) or double stranded nucleic acid (e.g., DNA that can integrate into the cell's genome or replicate episomally and that can.

SUMM [0734] Antisense And **Ribozyme** (Antagonists)

SUMM . . . the case of double stranded antisense nucleic acids, a single strand of the duplex DNA may thus be tested, or **triplex** formation may be assayed. The ability to hybridize will depend on both the degree of complementary and the length of. . . hybridizing nucleic acid, the more base mismatches with a RNA it may contain and still form a stable duplex (or **triplex** as the case may be). One skilled in the art can ascertain a tolerable degree of mismatch by use of. . .

SUMM [0748] Potential antagonists according to the invention also include catalytic RNA, or a **ribozyme** (See, e.g., PCT International Publication WO 90/11364, published Oct. 4, 1990; Sarver et al, Science 247:1222-1225 (1990). While **ribozymes** that cleave mRNA at site specific recognition sequences can be used to destroy mRNAs, the use of hammerhead **ribozymes** is preferred. Hammerhead **ribozymes** cleave mRNAs at locations dictated by flanking regions that form complementary base pairs with the target mRNA. The sole requirement is that the target mRNA have the following sequence of two bases: 5'-UG-3'. The construction and production of hammerhead **ribozymes** is well known in the art and is described more fully in Haseloff and Gerlach, Nature 334:585-591 (1988). There are numerous potential hammerhead **ribozyme** cleavage sites within the nucleotide sequence of SEQ ID NO:X. Preferably, the **ribozyme** is engineered so that the cleavage recognition site is located near the 5' end of the mRNA; i.e., to increase. . .

SUMM [0749] As in the antisense approach, the **ribozymes** of the invention can be composed of modified oligonucleotides (e.g. for improved stability, targeting, etc.) and should be delivered to cells which express in vivo. DNA constructs encoding the **ribozyme** may be introduced into the cell in the same manner as described above for the introduction of antisense encoding DNA. A preferred method of delivery involves using a DNA construct "encoding" the **ribozyme** under the control of a strong constitutive promoter, such as, for example, pol III or pol II promoter, so that transfected cells will produce sufficient quantities of the **ribozyme** to destroy endogenous messages and inhibit translation. Since **ribozymes**

unlike antisense molecules, are catalytic, a lower intracellular concentration is required for efficiency.

SUMM . . . by administering to a patient (a) an antisense molecule directed to the polynucleotide of the present invention, and/or (b) a **ribozyme** directed to the polynucleotide of the present invention.

L3 ANSWER 7 OF 24 USPATFULL

ACCESSION NUMBER: 2002:272900 USPATFULL
TITLE: Stimulus-inducible protein kinase complex and methods of use therefor
INVENTOR(S): Mercurio, Frank, San Diego, CA, UNITED STATES
Zhu, Hengyi, San Diego, CA, UNITED STATES
Barbosa, Miguel, San Diego, CA, UNITED STATES
Li, Jian Wu, San Diego, CA, UNITED STATES
Murray, Brion W., San Diego, CA, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002151021	A1	20021017
APPLICATION INFO.:	US 2001-844908	A1	20010427 (9)
RELATED APPLN. INFO.:	Division of Ser. No. US 1997-910820, filed on 13 Aug 1997, PATENTED Continuation-in-part of Ser. No. US 1996-697393, filed on 26 Aug 1996, PATENTED		
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	APPLICATION		
LEGAL REPRESENTATIVE:	Timothy X. Gibson, Mathews, Collins, Shepherd & Gould, Suite 306, 100 Thanet Circle, Princeton, NJ, 08540		
NUMBER OF CLAIMS:	32		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	31 Drawing Page(s)		
LINE COUNT:	2343		
CAS INDEXING IS AVAILABLE FOR THIS PATENT.			
DETD . . .	as "bait" in standard two-hybrid screens to identify other regulatory molecules, which may include IKK-1, IKK-2, NF.kappa.B1, RelA, I.kappa.B.beta. and/or p70 S6 kinase (Kieran et al., Cell 62:1007-1018, 1990; Nolan et al., Cell 64:961-69, 1991; Thompson et al., Cell 80:573-82, 1995; Grove et. . .		
DETD . . .	peptide that represents the substrate binding domain of I.kappa.B kinase or a phosphorylation motif of I.kappa.B, an antisense polynucleotide or ribozyme that interferes with transcription and/or translation of I.kappa.B kinase, a molecule that inactivates IKK signalsome by binding to the complex,. . .		

L3 ANSWER 8 OF 24 USPATFULL

ACCESSION NUMBER: 2002:258894 USPATFULL
TITLE: 38646, a novel guanine nucleotide exchange factor and uses therefor
INVENTOR(S): Glucksmann, Maria Alexandra, Lexington, MA, UNITED STATES
PATENT ASSIGNEE(S): Millennium Pharmaceuticals, Inc., Cambridge, MA (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002142464	A1	20021003
APPLICATION INFO.:	US 2001-950491	A1	20010910 (9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-231089P	20000908 (60)
DOCUMENT TYPE:	Utility	
FILE SEGMENT:	APPLICATION	
LEGAL REPRESENTATIVE:	AKIN, GUMP, STRAUSS, HAUER & FELD, L.L.P., ONE COMMERCE	

SQUARE, 2005 MARKET STREET, SUITE 2200, PHILADELPHIA,
PA, 19103

NUMBER OF CLAIMS: 22
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 15 Drawing Page(s)
LINE COUNT: 4625

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

SUMM . . . affect cytoskeletal organization. These proteins, which exhibit GTPase activity, are also known to be involved in activation of Jun amino-terminal **kinase**, regulation of **p70 S6 kinase**, and induction of transcriptional activation of genes containing the serum response element.

DETD [0128] Antisense Nucleic Acid Molecules, **Ribozymes** and Modified 38646 Nucleic Acid Molecules

DETD [0134] In still another embodiment, an antisense nucleic acid of the invention is a **ribozyme**. A **ribozyme** having specificity for a 38646-encoding nucleic acid can include one or more sequences complementary to the nucleotide sequence of a . . .

DETD . . . fragment length sizes between sample and control DNA indicates mutations in the sample DNA. Moreover, the use of sequence specific **ribozymes** (e.g., U.S. Pat. No. 5,498,531) can be used to score for the presence of specific mutations by development or loss of a **ribozyme** cleavage site.

DETD [0365] Further, antisense and **ribozyme** molecules that inhibit expression of the target gene can also be used in accordance with the invention to reduce the . . . target gene activity. Still further, triple helix molecules can be utilized in reducing the level of target gene activity. Antisense, **ribozyme** and triple helix molecules are discussed above.

DETD [0366] It is possible that the use of antisense, **ribozyme**, and/or triple helix molecules to reduce or inhibit mutant gene expression can also reduce or inhibit the transcription (triple helix) and/or translation (antisense, **ribozyme**) of mRNA produced by normal target gene all eles, such that the concentration of normal target gene product present can. . .

L3 ANSWER 9 OF 24 USPATFULL

ACCESSION NUMBER: 2002:251221 USPATFULL
TITLE: ASIP-related proteins
INVENTOR(S): Reddy, Roopa, Sunnyvale, CA, UNITED STATES
Tang, Y. Tom, San Jose, CA, UNITED STATES
Baughn, Mariah R., San Leandro, CA, UNITED STATES
Krasnow, Randi E., Stanford, CA, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002137166	A1	20020926
APPLICATION INFO.:	US 2001-757781	A1	20010109 (9)
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	APPLICATION		
LEGAL REPRESENTATIVE:	INCYTE GENOMICS, INC., 3160 PORTER DRIVE, PALO ALTO, CA, 94304		
NUMBER OF CLAIMS:	22		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	40 Drawing Page(s)		
LINE COUNT:	3608		

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

SUMM . . . tumor progression (Artagaveytia et al. (1997) J Steroid Biochem Mol Biol 60:221-228). EGF binds to its receptor, the protein tyrosine **kinase** EGF receptor (EGFR), which is also known as erbB2 (Carpenter (2000) Bioessays 22:697-707). Ligation of EGF to EGFR results in the activation of the tyrosine **kinase** domain of EGFR and the phosphorylation of multiple substrates. EGF may regulate multimeric signaling complexes associated with aPKCs. In cells. . . al. (1996)

EMBO J 15:788-798). EGF also influences the activity of PKC.zeta.. In response to EGF, PKC.zeta. phosphorylates and activates **p70 S6 kinase**, a regulator of cell proliferation (Romanelli et al. (1999) Mol Cell Biol 19:2921-2928).

DETD [0086] **Ribozymes**, enzymatic RNA molecules, may also be used to catalyze the specific cleavage of RNA. The mechanism of **ribozyme** action involves sequence-specific hybridization of the **ribozyme** molecule to complementary target RNA followed by endonucleolytic cleavage at sites such as GUA, GUU, and GUC. Once such sites. . .

DETD [0087] Complementary nucleic acids and **ribozymes** of the invention may be prepared via recombinant expression, in vitro or in vivo, or using solid phase phosphoramidite chemical. . .

L3 ANSWER 10 OF 24 USPATFULL

ACCESSION NUMBER: 2002:227650 USPATFULL
TITLE: Integrin-linked kinase and its uses
INVENTOR(S): Dedhar, Shoukat, Vancouver, CANADA
Hannigan, Greg, Toronto, CANADA

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002122801	A1	20020905
APPLICATION INFO.:	US 2001-840704	A1	20010423 (9)
RELATED APPLN. INFO.:	Continuation of Ser. No. US 2000-566906, filed on 9 May 2000, GRANTED, Pat. No. US 6369205 Division of Ser. No. US 1999-390425, filed on 3 Sep 1999, GRANTED, Pat. No. US 6338958 Continuation-in-part of Ser. No. US 1997-955841, filed on 21 Oct 1997, GRANTED, Pat. No. US 6013782 Continuation-in-part of Ser. No. US 1996-752345, filed on 19 Nov 1996, ABANDONED		

	NUMBER	DATE
PRIORITY INFORMATION:	US 1995-9074P	19951221 (60)
DOCUMENT TYPE:	Utility	
FILE SEGMENT:	APPLICATION	
LEGAL REPRESENTATIVE:	Pamela J. Sherwood, Bozicevic, Field & Francis LLP, Suite 200, 200 Middlefield Road, Menlo Park, CA, 94024	
NUMBER OF CLAIMS:	18	
EXEMPLARY CLAIM:	1	
NUMBER OF DRAWINGS:	23 Drawing Page(s)	
LINE COUNT:	3236	

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

DETD [0094] As an alternative to anti-sense inhibitors, catalytic nucleic acid compounds, e.g. **ribozymes**, anti-sense conjugates, etc. may be used to inhibit gene expression. **Ribozymes** may be synthesized in vitro and administered to the patient, or may be encoded on an expression vector, from which the **ribozyme** is synthesized in the targeted cell (for example, see International patent application WO 9523225, and Beigelman et al. (1995) Nucl.. . .

DETD [0207] Since Ptdlns(3,4,5,)P3 is specifically generated upon receptor-mediated stimulation of PI(3)**Kinase** activity, it was determined whether ILK activity is stimulated in a PI(3)K dependent manner. PI(3)K is activated in response to. . . second messenger that acts on pathways that control cell proliferation, cell survival, and metabolic changes often through the activation of **p70 ribosomal S6 Kinase (p70.sup.S6k)** and protein **kinase B (PKB)**, also known as **AKT**. PKB/AKT is a protooncogene and has been shown to be activated in a PI(3)K-dependent.

L3 ANSWER 11 OF 24 USPATFULL

ACCESSION NUMBER: 2002:72627 USPATFULL
TITLE: Nucleic, acids, proteins, and antibodies

INVENTOR(S) : Rosen, Craig A., Laytonsville, MD, UNITED STATES
 Ruben, Steven M., Olney, MD, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002039764	A1	20020404
APPLICATION INFO.:	US 2001-925298	A1	20010810 (9)
RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. WO 2000-US5881, filed on 8 Mar 2000, UNKNOWN		

	NUMBER	DATE	
PRIORITY INFORMATION:	US 1999-124270P	19990312 (60)	
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	APPLICATION		
LEGAL REPRESENTATIVE:	HUMAN GENOME SCIENCES INC, 9410 KEY WEST AVENUE, ROCKVILLE, MD, 20850		
NUMBER OF CLAIMS:	23		
EXEMPLARY CLAIM:	1		
LINE COUNT:	20087		
CAS INDEXING IS AVAILABLE FOR THIS PATENT.			
SUMM . . . 90	HHESJ29 [Homo sapiens] >sp G4100632 G4100632 LYMPHOID PHOSPHATASE LYP 1. Length = 808		
189	792931		
	1	576	HEGAW71
190	792943		myosin heavy chain kinase B [Dictyostelium
	gi 1903458		3 1247 43 68 HDPRZ79 discoideum]>sp P90648 KMH_B_DICDI MYOSIN HEAVY CHAIN KINASE B (BC 2.7.1.129) (MHCK B). Length = 732
191	793104		
	107	250	HKGAJ80
192	793445		desmoyokin - human (fragments)
	pir A45259 A45		1 723. . . >gi 337762 prosaposin [Homo
	sapiens]		>gi 337756 sphingolipid activator precursor [Homo sapiens] Length = 524
227	813262		
	1	345	HFKCA89
228	815637		(AC004003) serine/threonine kinase RICK;
	gi 3264574		3 461 92 92 HNHDS66 match to protein AF027706 (PID:g3123887) and mRNA AF027706 (NID:g3123886) [Homo sapiens] >gi 3290172 (AE064824) CARD-containing ICE associated kinase [Homo sapiens] >gi 3342910 (AF078530) receptor interacting prote calcyphosine [Homo sapiens] >gi 3075376 (AC004602) CAYP_HUMAN;. . . CALMODULIN. Length = 149
229	815853		
	gnl PID e24587		8 667 100 100 HLHAY85 (AC004602) CAYP_HUMAN;. . . CALMODULIN. Length = 149
241	827732		alternate name ygiG; ORF_f123 [Escherichia
	gi 882580		181 282 91 95 HBGDE81 coli] >gi 1789438 (AE000387) putative kinase [Escherichia coli] >pir H65093 H65093 ygiG protein - Escherichia coli (strain K- 12) >sp P31055 FOLB_ECOLI PROBABLE DHYDRONEOPTERIN ALDOLASE (EC 4.1.2.25) (DHNA). {SUB
242	827735		
	541	708	HHEDU22
243	827740		

716 838 HBNAP17
 244 827808
 86 1657 HMELR44
 245 828251 (AB016869) p70 ribosomal 56 kinase
 beta gnl|PID|d10353 134 949 91 91
 HNGOL64
 [Homo sapiens] >sp|D1035383|D1035383P70 83
 RIBOSOMAL S6 KINASE BETA. Length =
 495
 246 828357
 1 768 HKIYP61
 247 828449
 1 723 HBXCZ22
 248 828612 syntaxin 5 [Homo sapiens]
 gi|886071 68 460. . .
 SUMM . . . targeted cell. In another example, the invention provides a
 method for delivering a single stranded nucleic acid (e.g., antisense or
ribozymes) or double stranded nucleic acid (e.g., DNA that can
 integrate into the cell's genome or replicate episomally and that can.
 SUMM . . . cancer cell). In another example, the invention provides a
 method for delivering a single stranded nucleic acid (e.g., antisense or
ribozymes) or double stranded nucleic acid (e.g., DNA that can
 integrate into the cell's genome or replicate episomally and that can.
 SUMM [0519] Antagonists of the invention include, for example, binding and/or
 inhibitory antibodies, antisense nucleic acids, **ribozymes** or
 soluble forms of the polypeptides of the present invention (e.g., Fc
 fusion protein; see, e.g., Example 9). Agonists of. . .
 SUMM . . . targeted cell. In another example, the invention provides a
 method for delivering a single stranded nucleic acid (e.g., antisense or
ribozymes) or double stranded nucleic acid (e.g., DNA that can
 integrate into the cell's genome or replicate episomally and that can.
 SUMM [0746] Antisense And **Ribozyme** (Antagonists)
 SUMM . . . the case of double stranded antisense nucleic acids, a single
 strand of the duplex DNA may thus be tested, or **triplex**
 formation may be assayed. The ability to hybridize will depend on both
 the degree of complementarity and the length of. . . hybridizing
 nucleic acid, the more base mismatches with a RNA it may contain and
 still form a stable duplex (or **triplex** as the case may be).
 One skilled in the art can ascertain a tolerable degree of mismatch by
 use of.
 SUMM [0760] Potential antagonists according to the invention also include
 catalytic RNA, or a **ribozyme** (See, e.g., PCT International
 Publication WO 90/11364, published October 4, 1990; Sarver et al,
 Science 247:1222-1225 (1990). While **ribozymes** that cleave mRNA
 at site specific recognition sequences can be used to destroy mRNAs, the
 use of hammerhead **ribozymes** is preferred. Hammerhead
ribozymes cleave mRNAs at locations dictated by flanking regions
 that form complementary base pairs with the target mRNA. The sole
 requirement is that the target mRNA have the following sequence of two
 bases: 5'-UG-3'. The construction and production of hammerhead
ribozymes is well known in the art and is described more fully
 in Haseloff and Gerlach, Nature 334:585-591 (1988). There are numerous
 potential hammerhead **ribozyme** cleavage sites within the
 nucleotide sequence of SEQ ID NO:X. Preferably, the **ribozyme**
 is engineered so that the cleavage recognition site is located near the
 5' end of the mRNA; i.e., to increase. . .
 SUMM [0761] As in the antisense approach, the **ribozymes** of the
 invention can be composed of modified oligonucleotides (e.g. for
 improved stability, targeting, etc.) and should be delivered to cells
 which express in vivo. DNA constructs encoding the **ribozyme**
 may be introduced into the cell in the same manner as described above

for the introduction of antisense encoding DNA. A preferred method of delivery involves using a DNA construct "encoding" the **ribozyme** under the control of a strong constitutive promoter, such as, for example, pol III or pol II promoter, so that transfected cells will produce sufficient quantities of the **ribozyme** to destroy endogenous messages and inhibit translation. Since **ribozymes** unlike antisense molecules, are catalytic, a lower intracellular concentration is required for efficiency.

SUMM . . . by administering to a patient (a) an antisense molecule directed to the polynucleotide of the present invention, and/or (b) a **ribozyme** directed to the polynucleotide of the present invention.

L3 ANSWER 12 OF 24 USPATFULL

ACCESSION NUMBER: 2002:32181 USPATFULL
 TITLE: Methods of monitoring enzyme activity
 INVENTOR(S): Griffiths, Gary, Oldham, UNITED KINGDOM

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002019002	A1	20020214
APPLICATION INFO.:	US 2001-877919	A1	20010607 (9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-211313P	20000613 (60)
DOCUMENT TYPE:	Utility	
FILE SEGMENT:	APPLICATION	
LEGAL REPRESENTATIVE:	PALMER & DODGE, LLP, ONE BEACON STREET, BOSTON, MA, 02108-3190	
NUMBER OF CLAIMS:	40	
EXEMPLARY CLAIM:	1	
LINE COUNT:	3633	

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

SUMM [0138] Further examples of protein kinases identified to date include the protein tyrosine **kinase** subfamily (such as PDGF receptors, EGF receptors, src family kinases (see Brown & Cooper, 1996, Biochimica and Biophysica Acta 1287:121-149 for a review), the JAK **kinase** family (such as JAK1, JAK2 and tyk2), Erb B2, Bcr-Abl, Alk, Trk, Res/Sky for a detailed review see Al-Obeidi et al., 1998, Biopolymers (Peptide Science) Vol. 47:97-223), the MAP **kinase** pathway subfamily (such as the MAP family, the ERK family, the MEK family, the MEKK family, RAF-1 and JNK), the cyclin-dependent **kinase** subfamily (such as p.sub.34cdc2 and cdk2--see Nigg, 1995, Bioessays 17:471-480 for a review), Weel/Mytl, polo-like kinases (such as plkl, Plxl, POLO, Snk, Fnk/Prk Sak-a, Sak-b--see Lane & Nigg, 1997, Trends in Cell Biol. 7:63-68), the receptor serine **kinase** subfamily, protein **kinase** C (PK-C), cyclic-AMP dependent **kinase** (PK-A), cyclic-GMP dependent **kinase**, Ca.sup.2+/calmodulin dependent kinases (such as CaM **kinase** I, II and IV), DNA dependent protein **kinase**, phosphoinositide 3-kinases, PDK-1, the p21-activated protein **kinase** family (PAKs), such as Pak1, Pak2 and Pak3--see Sells & Chernoff, 1997, Trends in Cell Biol. 7:162-167), p70 S6 **kinase**, I.kappa.B **kinase**, casein **kinase** II, glycogen-synthase kinases.

SUMM . . . protozoal antigens and parasitic antigens. Candidate modulators additionally comprise proteins, lipoproteins, glycoproteins, phosphoproteins and nucleic acids (e.g., RNAs such as **ribozymes** or antisense nucleic acids). Proteins or polypeptides which can be screened using the methods of the present invention include hormones,.

SUMM . . . described above, but may eliminate or encode products which eliminate deleterious proteins. Such nucleic acid sequences are antisense RNA and **ribozymes**, as well as DNA expression

constructs that encode them. Note that antisense RNA molecules, **ribozymes** or genes encoding them may be administered to a test cell or organism by a method of nucleic acid delivery that is known in the art, as described below. Inactivating nucleic acid sequences may encode a **ribozyme** or antisense RNA specific for the a target mRNA. **Ribozymes** of the hammerhead class are the smallest known, and lend themselves both to in vitro production and delivery to cells. . . .

SUMM [0331] When the end product (e.g., an antisense RNA molecule or **ribozyme**) is administered directly, the dosage to be administered is directly proportional to the amount needed per cell and the number. . . .

L3 ANSWER 13 OF 24 USPATFULL

ACCESSION NUMBER: 2002:12284 USPATFULL
 TITLE: Arrayed transfection method and uses related thereto
 INVENTOR(S): Sabatini, David M., Cambridge, MA, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002006664	A1	20020117
APPLICATION INFO.:	US 2001-817003	A1	20010322 (9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-193580P	20000330 (60)
	US 1999-154737P	19990917 (60)
DOCUMENT TYPE:	Utility	
FILE SEGMENT:	APPLICATION	
LEGAL REPRESENTATIVE:	ROPES & GRAY, ONE INTERNATIONAL PLACE, BOSTON, MA, 02110-2624	
NUMBER OF CLAIMS:	37	
EXEMPLARY CLAIM:	1	
NUMBER OF DRAWINGS:	7 Drawing Page(s)	
LINE COUNT:	2671	

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

DETD . . . as a protein that confers drug resistance. Examples of heterologous RNA include, but are not limited to, anti-sense RNA sequences, **ribozymes**, and double-stranded RNA (for inducing sequence-specific RNA interference).

DETD . . . for a protein, a "coding" sequence for an RNA molecule (e.g., which is transcribed into an anti-sense RNA sequence, a **ribozyme** or double-stranded RNA), or a regulatory sequence (e.g., as part of a reporter construct), to name but a few examples.

DETD . . . SGEs that can be identified by the subject method include, but are not limited to, polypeptides, inhibitory antisense RNA molecules, **ribozymes**, nucleic acid decoys, and small peptides.

DETD . . . expressed as part of a fusion protein. In other embodiments the inserted fragment alone may be expressed. In another embodiment, **ribozyme**-encoding sequences may be inserted directly adjacent to the insert to allow for selection of most efficient **ribozyme**-antisense clones. In still other embodiments the gene suppression element library may be further modified by random mutagenesis procedures known in. . . .

DETD . . . SGEs that can be identified by the subject method include, but are not limited to, polypeptides, inhibitory antisense RNA molecules, **ribozymes**, nucleic acid decoys, and small peptides. For instance, a gene whose activity is inactivated by an identified SGE can itself. . . .

DETD . . . assaying the phenotype. These modifications could be derived from methods such as overexpression, knockout constructs, dominant negative mutants, anti-sense RNA, **ribozyme** RNA or others. The resulting phenotypic change could be assayed under different environmental conditions, genetic backgrounds and cell types. For. . . .

DETD	apoptosis	AF016266	TRAIL receptor 2
2:D10	cell adhesion	X97229	NK receptor
2:F7	cell adhesion	M98399	CD36
1:A9	nuclear	U11791	CyclinH
1:B5	nuclear	M60527	deoxycytidine
	kinase		
1:B12	nuclear	M60724	p70
	S6 kinase kinase .alpha.1		
1:C12	nuclear	M90813	D-type cyclin
1:E4	mitochondrial	U54645	methylnalonyl-coA
	mutase		
1:E10	mitochondrial	J05401	creatine kinase
1:G9	nuc/cyto	U40989	tat interactive protein
1:G10	nuc/cyto	U09578	MAPKAP (3pk)
	kinase		
2:A9	nuclear	X83928	TFIID subunit TAFII28
2:A12	nuc/cyto	M62831	.ETR101
2:B6	nuc/cyto	X06948	IgE receptor
	.alpha.-subunit		
2:B12	nuclear	X63469	TFIIE .beta. subunit
2:C5	nuclear	M76766	

L3 ANSWER 14 OF 24 USPATFULL

ACCESSION NUMBER: 2002:75564 USPATFULL
 TITLE: Integrin-linked kinase and its uses
 INVENTOR(S): Dedhar, Shoukat, Vancouver, CANADA
 Hannigan, Greg, Ontario, CANADA
 PATENT ASSIGNEE(S): Sunnybrook Health Science Centre, Toronto, CANADA
 (non-U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6369205	B1	20020409
APPLICATION INFO.:	US 2000-566906		20000509 (9)
RELATED APPLN. INFO.:	Division of Ser. No. US 1999-390425, filed on 3 Sep 1999 Continuation-in-part of Ser. No. US 1997-955841, filed on 21 Oct 1997, now patented, Pat. No. US 6013782 Continuation-in-part of Ser. No. US 1996-752345, filed on 19 Nov 1996, now abandoned		

	NUMBER	DATE
PRIORITY INFORMATION:	US 1995-9074P	19951221 (60)
DOCUMENT TYPE:	Utility	
FILE SEGMENT:	GRANTED	
PRIMARY EXAMINER:	Clark, Deborah J. R.	
ASSISTANT EXAMINER:	Chen, Shin-Lin	
LEGAL REPRESENTATIVE:	Sherwood, Pamela J., Bozicevic, Field & Francis LLP	
NUMBER OF CLAIMS:	5	
EXEMPLARY CLAIM:	1	
NUMBER OF DRAWINGS:	25 Drawing Figure(s); 23 Drawing Page(s)	
LINE COUNT:	3200	

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

DETD As an alternative to anti-sense inhibitors, catalytic nucleic acid compounds, e.g. **ribozymes**, anti-sense conjugates, etc. may be used to inhibit gene expression. **Ribozymes** may be synthesized in vitro and administered to the patient, or may be encoded on an expression vector, from which the **ribozyme** is synthesized in the targeted cell (for example, see International patent application WO 9523225, and Beigelman et al. (1995) Nucl. . . .

DETD Since Ptdlns(3,4,5)P3 is specifically generated upon receptor-mediated stimulation of PI(3)**Kinase** activity, it was determined whether ILK activity is stimulated in a PI(3)K dependent manner. PI(3)K is activated in response to. . . second messenger that acts on pathways

that control cell proliferation, cell survival, and metabolic changes often through the activation of **P70** ribosomal **S6 Kinase** (p.sub.70.sup.S6k) and protein **kinase B** (PKB), also known as AKT. PKB/AKT is a protooncogene and has been shown to be activated in a PI(3)K-dependent. . .

L3 ANSWER 15 OF 24 USPATFULL

ACCESSION NUMBER: 2002:9751 USPATFULL
 TITLE: Integrin-linked kinase and its uses
 INVENTOR(S): Dedhar, Shoukat, Vancouver, CANADA
 Hannigan, Greg, Ontario, CANADA
 PATENT ASSIGNEE(S): Sunnybrook Health Science Centre, Toronto, CANADA
 (non-U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6338958	B1	20020115
APPLICATION INFO.:	US 1999-390425		19990903 (9)
RELATED APPLN. INFO.:	Continuation of Ser. No. US 1998-35706, filed on 5 Mar 1998, now patented, Pat. No. US 6001622		
	Continuation-in-part of Ser. No. US 1997-955841, filed on 21 Oct 1997, now patented, Pat. No. US 6013782		
	Continuation-in-part of Ser. No. US 1996-752345, filed on 19 Nov 1996, now abandoned		

	NUMBER	DATE
PRIORITY INFORMATION:	US 1995-9074P	19951221 (60)
DOCUMENT TYPE:	Utility	
FILE SEGMENT:	GRANTED	
PRIMARY EXAMINER:	Clark, Deborah J. R.	
ASSISTANT EXAMINER:	Chen, Shin-Lin	
LEGAL REPRESENTATIVE:	Sherwood, Pamela J., Bozicevic, Field & Francis LLP	
NUMBER OF CLAIMS:	4	
EXEMPLARY CLAIM:	1	
NUMBER OF DRAWINGS:	21 Drawing Figure(s); 23 Drawing Page(s)	
LINE COUNT:	3203	

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

DETD As an alternative to anti-sense inhibitors, catalytic nucleic acid compounds, e.g. **ribozymes**, anti-sense conjugates, etc. may be used to inhibit gene expression. **Ribozymes** may be synthesized in vitro and administered to the patient, or may be encoded on an expression vector, from which the **ribozyme** is synthesized in the targeted cell (for example, see International patent application WO 9523225, and Beigelman et al. (1995) Nucl.. . .

DETD Since Ptdlns(3,4,5,)P3 is specifically generated upon receptor-mediated stimulation of PI(3)**Kinase** activity, it was determined whether ILK activity is stimulated in a PI(3)K dependent manner. PI(3)K is activated in response to. . . second messenger that acts on pathways that control cell proliferation, cell survival, and metabolic changes often through the activation of **P70** ribosomal **S6 Kinase** (**P70**.sup.S6k) and protein **kinase B** (PKB), also known as AKT. PKB/AKT is a protooncogene and has been shown to be activated in a PI(3)K-dependent. . .

L3 ANSWER 16 OF 24 USPATFULL

ACCESSION NUMBER: 2001:116764 USPATFULL
 TITLE: Ataxia-telangiectasia gene and its genomic organization
 INVENTOR(S): Shiloh, Yosef, Tel Aviv, Israel
 PATENT ASSIGNEE(S): Ramot-University Authority for Applied Research and Industrial Development, Tel Aviv, Israel (non-U.S. corporation)

NUMBER	KIND	DATE
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PATENT INFORMATION: US 6265158 B1 20010724
 WO 9636691 19961121

APPLICATION INFO.: US 1998-952014 19980202 (8)
 WO 1996-US7025 19960516
 19980202 PCT 371 date
 19980202 PCT 102(e) date

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 1996-629001, filed
 on 8 Apr 1996, now patented, Pat. No. US 5858661
 Continuation-in-part of Ser. No. US 1995-441822, filed
 on 16 May 1995, now patented, Pat. No. US 5756288

DOCUMENT TYPE: Utility
 FILE SEGMENT: GRANTED
 PRIMARY EXAMINER: Jones, W. Gary
 ASSISTANT EXAMINER: Goldberg, Jeanine
 LEGAL REPRESENTATIVE: Kohn & Associates
 NUMBER OF CLAIMS: 7
 EXEMPLARY CLAIM: 1,7
 NUMBER OF DRAWINGS: 4 Drawing Figure(s); 2 Drawing Page(s)
 LINE COUNT: 3109

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

DETD Recombinant methods known in the art can also be used to achieve the
 sense, antisense or **triplex** inhibition of a target nucleic
 acid. For example, vectors containing antisense nucleic acids can be
 employed to express protein or. . .

DETD Brown et al., "Control of **p70 S6 kinase** by
kinase activity of FRAP in vivo" Nature 377:441-446 (1995).

L3 ANSWER 17 OF 24 USPATFULL

ACCESSION NUMBER: 2001:107664 USPATFULL
 TITLE: Stimulus-inducible protein kinase complex and methods
 of use therefor
 INVENTOR(S): Mercurio, Frank, San Diego, CA, United States
 Zhu, Hengyi, San Diego, CA, United States
 Barbosa, Miguel, San Diego, CA, United States
 Li, Jian Wu, San Diego, CA, United States
 Murray, Brion W., San Diego, CA, United States
 PATENT ASSIGNEE(S): Signal Pharmaceuticals, Inc., San Diego, CA, United
 States (U.S. corporation)

	NUMBER	KIND	DATE

PATENT INFORMATION:	US 6258579	B1	20010710
APPLICATION INFO.:	US 1997-910820		19970813 (8)
RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 1996-697393, filed on 26 Aug 1996, now patented, Pat. No. US 5972674		
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	GRANTED		
PRIMARY EXAMINER:	Patterson, Jr., Charles L.		
LEGAL REPRESENTATIVE:	Mathews, Collins, Shepherd & Gould, P.A.		
NUMBER OF CLAIMS:	4		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	30 Drawing Figure(s); 28 Drawing Page(s)		
LINE COUNT:	1713		
CAS INDEXING IS AVAILABLE FOR THIS PATENT.			
DETD	. . . as "bait" in standard two-hybrid screens to identify other regulatory molecules, which may include IKK-1, IKK-2, NF.kappa.B1, RelA, I.kappa.B.beta. and/or p70 S6 kinase (Kieran et al., Cell 62:1007-1018, 1990; Nolan et al., Cell 64:961-69, 1991; Thompson et al., Cell 80:573-82, 1995; Grove et. . .		
DETD	. . . peptide that represents the substrate binding domain of I.kappa.B kinase or a phosphorylation motif of I.kappa.B, an antisense polynucleotide or ribozyme that interferes with transcription and/or translation of I.kappa.B kinase, a molecule that inactivates IKK		

signalsome by binding to the complex, . . .

L3 ANSWER 18 OF 24 USPATFULL

ACCESSION NUMBER: 2001:48208 USPATFULL
TITLE: Ataxia-telangiectasia gene
INVENTOR(S): Shiloh, Yosef, Tel Aviv, Israel
Tagle, Danilo A., Gaithersburg, MD, United States
Collins, Francis, Rockville, MD, United States
PATENT ASSIGNEE(S): The United States of America as represented by the
Department of Health and Human Services, Washington,
DC, United States (U.S. government)
Ramat University Authority for Applied Research and
Industrial Dev., Israel (non-U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6211336	B1	20010403
	WO 9636695		19961121
APPLICATION INFO.:	US 1998-952127		19980226 (8)
	WO 1996-US7040		19960516
			19980226 PCT 371 date
			19980226 PCT 102(e) date
RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 1995-508836, filed on 28 Jul 1995, now patented, Pat. No. US 5777093 Continuation-in-part of Ser. No. US 1995-493092, filed on 21 Jun 1995, now patented, Pat. No. US 5728807 Continuation-in-part of Ser. No. US 1995-441822, filed on 16 May 1995, now patented, Pat. No. US 5756288		
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	Granted		
PRIMARY EXAMINER:	Cintins, Marianne M.		
ASSISTANT EXAMINER:	Delacroix-Muirheid, C.		
LEGAL REPRESENTATIVE:	Kohn & Associates		
NUMBER OF CLAIMS:	5		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	10 Drawing Figure(s); 4 Drawing Page(s)		
LINE COUNT:	2279		
CAS INDEXING IS AVAILABLE FOR THIS PATENT.			
DETD	Recombinant methods known in the art can also be used to achieve the sense, antisense or triplex inhibition of a target nucleic acid. For example, vectors containing antisense nucleic acids can be employed to express protein or. . .		
DETD	Brown et al., "Control of p70 S6 kinase by kinase activity of FRAP in vivo" Nature 377:441-446 (1995).		

L3 ANSWER 19 OF 24 USPATFULL

ACCESSION NUMBER: 2001:36598 USPATFULL
TITLE: Mutated forms of the ataxia-telangiectasia gene and
method to screen for a partial A-T phenotype
INVENTOR(S): Shiloh, Yosef, Tel Aviv, Israel
PATENT ASSIGNEE(S): Ramot-University Authority for Applied Research and
Industrial Development Ltd., Tel Aviv, Israel (non-U.S.
corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6200749	B1	20010313
APPLICATION INFO.:	US 1996-642274		19960503 (8)
RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 1996-629001, filed on 8 Apr 1996 Continuation-in-part of Ser. No. US 1995-441822, filed on 16 May 1995, now patented, Pat. No. US 5756288		
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	Granted		

PRIMARY EXAMINER: Arthur, Lisa B.
LEGAL REPRESENTATIVE: Kohn & Associates
NUMBER OF CLAIMS: 5
EXEMPLARY CLAIM: 1,4
NUMBER OF DRAWINGS: 8 Drawing Figure(s); 2 Drawing Page(s)
LINE COUNT: 3090

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

DETD Recombinant methods known in the art can also be used to achieve the sense, antisense or **triplex** inhibition of a target nucleic acid. For example, vectors containing antisense nucleic acids can be employed to express protein or. . .

DETD Brown et al., "Control of **p70 S6 kinase** by **kinase** activity of FRAP in vivo" Nature 377:441-446 (1995).

L3 ANSWER 20 OF 24 USPATFULL

ACCESSION NUMBER: 2000:164282 USPATFULL
TITLE: Serine/threonine protein kinases
INVENTOR(S): Bandman, Olga, Mountain View, CA, United States
Tang, Y. Tom, San Jose, CA, United States
Goli, Surya K., San Jose, CA, United States
Corley, Neil C., Mountain View, CA, United States
Guegler, Karl J., Menlo Park, CA, United States
Gorgone, Gina A., Boulder Creek, CA, United States
Hillman, Jennifer L., Mountain View, CA, United States
PATENT ASSIGNEE(S): Incyte Pharmaceuticals, Inc., Palo Alto, CA, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6156523		20001205
APPLICATION INFO.:	US 1998-153939		19980916 (9)
RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 1996-749902, filed on 15 Nov 1996, now patented, Pat. No. US 5985635		
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	Granted		
PRIMARY EXAMINER:	Mertz, Prema		
ASSISTANT EXAMINER:	Murphy, Joseph F.		
LEGAL REPRESENTATIVE:	Incyte Pharmaceuticals, Inc., Murry, Lynn E.		
NUMBER OF CLAIMS:	5		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	14 Drawing Figure(s); 14 Drawing Page(s)		
LINE COUNT:	2733		

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

SUMM . . . different substrate specificities and responds to distinct extracellular stimuli (Egan, S. E. and Weinberg, R. A. (1993) Nature 365:781-783). MAP **kinase** signaling pathways are present in mammalian cells as well as in yeast. The extracellular stimuli which activate mammalian pathways include. . . cytokines such as tumor necrosis factor (TNF) and interleukin-1 (IL-1). An important member of the MAP kinases is the cytoplasmic **p70 ribosomal S6 kinase** which is essential for the initiation of protein synthesis in all cell types following mitogenic stimulation (Hershey, J. W. B. (1989) J. Biol. Chem. 264: 20823-26). Altered MAP **kinase** expression can therefore be implicated in a variety of disease conditions including cancer, inflammation, immune disorders, and disorders affecting growth. . .

DETD . . . the double helix to open sufficiently for the binding of polymerases, transcription factors, or regulatory molecules. Recent therapeutic advances using **triplex** DNA have been described in the literature. (See, e.g., Gee, J. E. et al. (1994) in Huber, B. E. and. . .

DETD **Ribozymes**, enzymatic RNA molecules, may also be used to catalyze the specific cleavage of RNA. The mechanism of **ribozyme** action involves sequence-specific hybridization of the **ribozyme**

molecule to complementary target RNA, followed by endonucleolytic cleavage. For example, engineered hammerhead motif **ribozyme** molecules may specifically and efficiently catalyze endonucleolytic cleavage of sequences encoding HSTK.

DETD Specific **ribozyme** cleavage sites within any potential RNA target are initially identified by scanning the target molecule for **ribozyme** cleavage sites, including the following sequences: GUA, GUU, and GUC. Once identified, short RNA sequences of between 15 and 20.

DETD Complementary ribonucleic acid molecules and **ribozymes** of the invention may be prepared by any method known in the art for the synthesis of nucleic acid molecules.. . .

L3 ANSWER 21 OF 24 USPATFULL

ACCESSION NUMBER: 1999:163477 USPATFULL
TITLE: Integrin-linked kinase and its use
INVENTOR(S): Dedhar, Shoukat, Vancouver, Canada
Hannigan, Greg, Ontario, Canada
PATENT ASSIGNEE(S): Sunnybrook Health Science Centre, Ontario, Canada
(non-U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6001622		19991214
APPLICATION INFO.:	US 1998-35706		19980305 (9)
RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 1997-955841, filed on 21 Oct 1997 which is a continuation-in-part of Ser. No. US 1996-752345, filed on 19 Nov 1996		

	NUMBER	DATE
PRIORITY INFORMATION:	US 1995-9074P	19951221 (60)
DOCUMENT TYPE:	Utility	
FILE SEGMENT:	Granted	
PRIMARY EXAMINER:	Achutamurthy, Ponnathapu	
LEGAL REPRESENTATIVE:	Bozicevic, Field & Francis LLP, Sherwood, Pamela	
NUMBER OF CLAIMS:	4	
EXEMPLARY CLAIM:	1	
NUMBER OF DRAWINGS:	21 Drawing Figure(s); 23 Drawing Page(s)	
LINE COUNT:	3148	

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

DETD As an alternative to anti-sense inhibitors, catalytic nucleic acid compounds, e.g. **ribozymes**, anti-sense conjugates, etc. may be used to inhibit gene expression. **Ribozymes** may be synthesized in vitro and administered to the patient, or may be encoded on an expression vector, from which the **ribozyme** is synthesized in the targeted cell (for example, see International patent application WO 9523225, and Beigelman et al. (1995) Nucl.. . .

DETD Since Ptdlns(3,4,5,)P3 is specifically generated upon receptor-mediated stimulation of PI(3)**Kinase** activity, it was determined whether ILK activity is stimulated in a PI(3)K dependent manner. PI(3)K is activated in response to. . . second messenger that acts on pathways that control cell proliferation, cell survival, and metabolic changes often through the activation of **P70 ribosomal S6 Kinase (p70.sup.S6k)** and protein **kinase B** (PKB), also known as AKT. PKB/AKT is a protooncogene and has been shown to be activated in a PI(3)K-dependent. . .

L3 ANSWER 22 OF 24 USPATFULL

ACCESSION NUMBER: 1999:146335 USPATFULL
TITLE: Nucleic acids encoding novel human serine/threonine protein kinases
INVENTOR(S): Bandman, Olga, Mountain View, CA, United States
Goli, Surya K., Sunnyvale, CA, United States

PATENT ASSIGNEE(S): Hillman, Jennifer L., San Jose, CA, United States
 Incyte Pharmaceuticals, Inc., Palo Alto, CA, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5985635		19991116
APPLICATION INFO.:	US 1996-749902		19961115 (8)
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	Granted		
PRIMARY EXAMINER:	Teng, Sally P.		
LEGAL REPRESENTATIVE:	Incyte Pharmaceuticals, Inc.		
NUMBER OF CLAIMS:	6		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	12 Drawing Figure(s); 12 Drawing Page(s)		
LINE COUNT:	2520		

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

SUMM . . . different substrate specificities and responds to distinct extracellular stimuli (Egan, S. E. and Weinberg, R. A. (1993) Nature 365:781-783). MAP **kinase** signaling pathways are present in mammalian cells as well as in yeast. The extracellular stimuli which activate mammalian pathways include. . . such as tumor necrosis factor (TNF) and interleukin- 1 (IL-1). An important member of the MAP kinases is the cytoplasmic **p70 ribosomal S6 kinase** which is essential for the initiation of protein synthesis in all cell types following mitogenic stimulation (Hershey, J.W.B. (1989) J. Biol. Chem. 264:20823-26). Altered MAP **kinase** expression can therefore be implicated in a variety of disease conditions including cancer, inflammation, immune disorders, and disorders affecting growth. . .

SUMM . . . and HSTK-2 and referred to collectively as HSTK (human serine/threonine kinases) characterized as having chemical and structural homology to two **p70 S6 ribosomal kinases** from man (GI 189508) and rabbit (GI 1562), and to a serine/threonine **kinase** from fetal liver (GI 1480861), and XEEK1, (GI 1016551), from the African frog, *Xenopus laevis*.

DRWD FIGS. 4A and 4B show the amino acid alignments between HSTK-2 (SEQ ID NO:3), the human **p70 S6**, (GI 189508: SEQ ID NO:7), and the rabbit **p70 S6 kinase** (GI 1562; SEQ ID NO:8).

DETD . . . polypeptide sequences disclosed herein is based in part on the chemical and structural homology among the novel HSTK and two **p70 S6 ribosomal kinases** from man (GI 189508) and rabbit (GI 1562), and a serine/threonine **kinase** from fetal liver (GI 1480861), and XEEK1, (GI 1016551) from the african frog. Because of the widespread roles for protein. . .

DETD . . . the double helix to open sufficiently for the binding of polymerases, transcription factors, or regulatory molecules. Recent therapeutic advances using **triplex** DNA have been described in the literature (Gee, J. E. et al. (1994) In: Huber, B. E. and B. I. . .

DETD **Ribozymes**, enzymatic RNA molecules, may also be used to catalyze the specific cleavage of RNA. The mechanism of **ribozyme** action involves sequence-specific hybridization of the **ribozyme** molecule to complementary target RNA, followed by endonucleolytic cleavage. Examples which may be used include engineered hammerhead motif **ribozyme** molecules that can specifically and efficiently catalyze endonucleolytic cleavage of sequences encoding HSTK.

DETD Specific **ribozyme** cleavage sites within any potential RNA target are initially identified by scanning the target molecule for **ribozyme** cleavage sites which include the following sequences: GUA, GUU, and GUC. Once identified, short RNA sequences of between 15 and. . .

DETD Antisense molecules and **ribozymes** of the invention may be

prepared by any method known in the art for the synthesis of RNA molecules. These.

DETD . . . protein substrate using gamma-labeled .sup.32 P-ATP and quantitation of the incorporated radioactivity using a gamma radioisotope counter. For identification of **p70 S6** ribosomal **kinase** activity, specifically, purified 40 S ribosomal protein is used as substrate (Flotow, H. and Thomas, G. (1992) J. Biol. Chem. 267:3074-78). HSTK is incubated with the protein substrate, .sup.32 P-ATP, and a **kinase** buffer. The .sup.32 P incorporated into the substrate is then separated from free .sup.32 P-ATP by electrophoresis and the incorporated.

L3 ANSWER 23 OF 24 USPATFULL

ACCESSION NUMBER: 1999:132563 USPATFULL
TITLE: Stimulus-inducible protein kinase complex and methods of use therefor
INVENTOR(S): Mercurio, Frank, San Diego, CA, United States
Zhu, Hengyi, San Diego, CA, United States
Barbosa, Miguel, San Diego, CA, United States
PATENT ASSIGNEE(S): Signal Pharmaceuticals, Inc., San Diego, CA, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5972674		19991026
APPLICATION INFO.:	US 1996-697393		19960826 (8)
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	Granted		
PRIMARY EXAMINER:	Chan, Christina Y.		
ASSISTANT EXAMINER:	Nolan, Patrick J.		
LEGAL REPRESENTATIVE:	SEED and BERRY LLP		
NUMBER OF CLAIMS:	2		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	11 Drawing Figure(s); 6 Drawing Page(s)		
LINE COUNT:	945		

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

DETD Components of a I.kappa.B.alpha. **kinase** complex may also be identified by performing any of a variety of protein-protein interaction assays known to those of ordinary. . . be used as "bait" in standard two-hybrid screens to identify other regulatory molecules such as, perhaps, NF.kappa.B1, RelA, I.kappa.B.beta. and/or **p70 S6 kinase** (Kieran et al., Cell 62:1007-1018, 1990; Nolan et al., Cell 64:961-969, 1991; Thompson et al., Cell 80:573-582, 1995; Grove et. . .

DETD . . . peptide that represents the substrate binding domain of I.kappa.B.alpha. kinase or the phosphorylation motif of I.kappa.B.alpha., an antisense polynucleotide or **ribozyme** that interferes with transcription and/or translation of I.kappa.B.alpha. kinase, a molecule that inactivates I.kappa.B.alpha. kinase complex by binding to the. . .

L3 ANSWER 24 OF 24 USPATFULL

ACCESSION NUMBER: 1999:4329 USPATFULL
TITLE: Ataxia-telangiectasia gene and its genomic organization
INVENTOR(S): Shiloh, Yosef, Tel Aviv, Israel
PATENT ASSIGNEE(S): RAMOT-University Authority for Applied Research and Industrial Development, Tel Aviv, Israel (non-U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5858661		19990112
APPLICATION INFO.:	US 1996-629001		19960408 (8)
RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 1995-441822, filed		

on 16 May 1995, now patented, Pat. No. US 5756288
DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Arthur, Lisa B.
LEGAL REPRESENTATIVE: Kohn & Associates
NUMBER OF CLAIMS: 9
EXEMPLARY CLAIM: 1,7
NUMBER OF DRAWINGS: 6 Drawing Figure(s); 2 Drawing Page(s)
LINE COUNT: 3461

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

DETD Recombinant methods known in the art can also be used to achieve the sense, antisense or **triplex** inhibition of a target nucleic acid. For example, vectors containing antisense nucleic acids can be employed to express protein or. . .

DETD Brown et al., "Control of **p70 S6 kinase** by **kinase** activity of FRAP in vivo" Nature 377:441-446 (1995).